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VARICOSE VEINS AND ULCERS: THEIR ÆTIOLOGY AND TREATMENT.[1]

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THE subject of varicose veins and their treatment is a fascinating one. Mention of the subject is found in the writings of Hippocrates, and ever since then various writers have commented on the condition, and numerous weird and wonderful theories have been advanced to account for the formation of these veins. But little real advance was made until about two decades ago, and general interest in the subject dates back to less than one decade. Since then, however, the modern injection technique has become very popular and we have had a simple curative measure placed at the disposal of any medical man who can wield a syringe. The method has caught on, but, as so often happens, although the technique of injection has been fairly well grasped, the underlying principles and some of the indications and possibilities are not so generally understood, and the object of my paper is to try to bring a better understanding of these features to you. There is not sufficient time to deal with the mass of detail which has accumulated on this subject, my object being simply to deal with broad principles and to stress the important points.

A varicose vein is one which has become dilated, elongated and tortuous, and in which the valves have become incompetent. Numerous changes take place in the wall, usually causing a considerable degree of thickening at first; but at a later stage we frequently find pouches in which the vein wall and skin together seem no thicker than tissue paper.

[1] Read at a meeting of the South Australian Branch of the British Medical Association on February 23, 1933.

In these later stages the vein is often adherent to the skin, which also is degenerated and atrophic. The degeneration affects all three coats of the vein wall, and the main changes are connective tissue proliferation and multiplication of the elastic fibres. The final result may be an almost complete disappearance of the muscular fibres.

ÆTIOLOGY.

Numerous theories have been brought forward to try to account for the histological appearance, and various observers have attributed them to toxic, mechanical, inflammatory or atrophic changes. But there is as yet no general agreement, and the matter must be considered as not yet settled.

If we proceed to consideration of the clinical investigations, however, we find more uniformity of opinion as to the cause of varicose veins. I think everyone agrees that heredity plays a big part. The familial character of the condition is well known, and sometimes we see the same segment of the same vein being the only one affected in members of the same family. But there is a difference of opinion as to whether the primary factor is an inherent weakness and deficiency of the valves, or an inherent weakness of the vein wall itself.

Although it cannot be taken as definitely settled, a series of experiments carried out by Turner Warwick suggests that inadequacy or absence of the valves, most often affecting those in the perforating or communicating veins, is the primary lesion. However, there is still a good deal of support for the view that, sometimes at any rate, the valvular incompetence is secondary to an acquired weakness of the walls, such weakness being due to some toxic or endocrine disturbance. If this be so, the sclerosing of the dilated segments of the vein would be followed by further degeneration of other veins (due to the continued action of the toxin or gland) with formation of new varicosities.

But whatever the cause, varices are most common at the spot where large trunks communicate through the muscles with the deep veins, that is, varices seem to start most commonly opposite the mouths of perforating veins.

There are several exciting factors which, given an inherent valvular weakness, may play a prominent part in the actual development of the varicosity, such as:

1. Employment necessitating long periods of standing.

2. Any obstruction of the venous return, such as pelvic tumour, thrombosis of femoral or iliac veins, mitral stenosis, cirrhosis of the liver *et cetera*.

3. Any cause of abdominal straining, for example, constipation, an enlarged prostate, stricture of the urethra.

4. Continued violent exertion, as in some forms of athletics.

5. Any condition tending to produce weakening of the vein wall (possibly through some action on its muscle fibres), such as a long illness or a pregnancy. The varicose condition often arises during the recovery from a severe illness, and we have all seen cases in which varicose veins have first formed during a pregnancy or during which, if present beforehand, they have become larger. The cause of this is uncertain, but as it comes on before the uterus is bulky enough to cause pressure, it is usually attributed to toxæmia, possibly of placental origin. With a mild degree of inherent valvular incompetence, this is sufficient to produce varicose veins during the pregnancy; but after the toxic effect has passed off, the vein walls may recover sufficiently for the valves to function adequately again. In a more marked degree of valvular incompetence the toxic effect of one pregnancy may produce a lesion from which the vein never recovers. These cases are quoted in favour of the primary weak wall with secondary valvular incompetence theory.

Many people, doubtless, have a mild degree of inherent valvular weakness, but never develop varicosities because no exciting factor enters their lives for a sufficient period to exert a deleterious effect.

There are three common sites in which varicose veins develop: (i) the leg, (ii) the rectum, giving rise to hæmorrhoids, (iii) the spermatic cord, giving rise to a varicocele. They sometimes, however, form elsewhere, notably in the vulva and the broad ligament. In this paper our attention is being confined to those occurring in the leg.

To understand the hydrostatics (or, should we say, hæmostatics) in a varicose venous system, we must first consider the normal arrangement. This, briefly, is divided into two systems, (*a*) the superficial and (*b*) the deep, each having a free communication with the other by means of perforating veins. The terminal portion of the internal saphenous vein, where it joins the femoral, and the terminal portion of the external saphenous vein, where it joins the popliteal, may be considered as the perforating veins.

And now we come to an important point. The valves of the perforating veins are so arranged that blood can flow only from the superficial to the deep system.

The main factors responsible for the venous return are:

1. Abdomino-thoracic suction. The rhythmic alterations of pressure in the thoracic and abdominal cavities exert a degree of suction highly favourable to the return of blood from the lower abdominal, iliac and femoral veins. When the thoracic and abdominal pressures are raised, however, as in violent straining, the suction is replaced by a positive back pressure along the veins and considerable force is required to drive the blood from the limbs along the compressed iliac veins.

2. Skeletal muscular contraction and relaxation, in combination with an adequate valve mechanism and tone of the vein wall. The muscle veins, the deep veins below the knee, and, to a less extent, the femoral vein, are all more or less closely valved and run in places subjected to the effect of muscular pressure. When the pressure is raised, as during

muscular contraction, the blood is forced out of the veins and, because of an adequate valve mechanism, it can travel only towards the heart. During muscular relaxation the pressure in these veins is much lowered and they are refilled from two sources, (*a*) from the venules and capillaries of the deep circulation and (*b*) from the superficial veins, *viâ* the perforating veins. This blood is then driven up the leg at the next contraction.

3. The *vis a tergo*. The *vis a tergo* is the force with which the blood enters the vein radicles from the capillaries. It is a continually acting force and during the muscular relaxation it forces the blood into the small veins, past the first valves, when it comes under the influence of muscular contraction.

Thus in the normal leg, during active movements, the *vis a tergo* forces the blood into the small veins beyond the first valves, skeletal muscular contraction forces it up the limb, and abdomino-thoracic suction plays a part as it nears the upper end. The superficial system is emptied into the deep *viâ* the perforating veins.

When the position of attention, with the muscles tonically contracted, is adopted, the only two factors acting are abdomino-thoracic suction and *vis a tergo*. The rate of return is much slowed, and the veins become distended and the leg becomes uncomfortable. But the point I wish to make clear is that these two factors can exert sufficient force to return the blood, even if the rate of return is not all that it might be. (Compare a completely paralysed leg or the leg of an anæsthetized person.)

Now let us see the effect of valvular incompetence of a perforating vein. During muscular contraction a squirt of blood is forced along the vein into the superficial system, and the blood's further progress is impeded by a column of blood above and a valvular mechanism below. Eventually this results in dilatation and thickening of the superficial vein wall, and the valves in the dilated part become incompetent. This throws more weight on the next valve, which eventually gives way, and the condition may become progressive. If it be the terminal valve of the internal saphenous which first gives way, this segment of the superficial system becomes exposed to the full pressure in the femoral vein, the next valve gives way, and so on, until the valvular mechanism of the whole internal saphenous system is broken down from groin to ankle, and in the standing position the whole system is kept full. But it needs to be emphasized that a varicosity can start opposite any perforating vein.

Occasionally the varicosity seems to start in the deep system and is followed by incompetence of valves of the perforating veins, and this is followed by an extensive superficial varicosity.

Let us now consider the venous return with a superficial system of varicose veins.

The *vis a tergo* and abdomino-thoracic suction are still acting as in the normal leg, and we have seen that these two factors are capable of keeping up a more or less adequate venous return, and in the varicose leg, although the rate is slower, an upward flow of blood is maintained in the standing, inactive position. Muscular contraction has little helpful effect—the veins are filled up by the contraction and emptied by the relaxation, and it is difficult to estimate whether the net result of a series of contractions and relaxations will be toward filling or emptying the vein. Thus in the standing inactive position, without any abdominal straining, the net result is a slow heartward flow.

If, however, a varicose leg is raised until the superficial veins are emptied and then lowered, it is easy to see the veins filling from above downwards. Also, when there is any increase in abdominal pressure, as in standing, the force required to empty the deep veins is much increased and blood overflows into the superficial system and may flow downwards to the next perforating vein, where it again enters the deep system and is forced up again.

Barber, however, states that the vicious circle theory is untenable; but whether it is or not, there is no doubt that the heartward flow along the veins is much reduced.

The pressure in varicose veins may sometimes be very high, especially if the femoral valve (that is, in the situation where the femoral vein becomes the iliac) is also incompetent. Then the varicose venous system becomes exposed to changes in intra-abdominal pressure, and readings as high as 260 millimetres of mercury have been produced by full straining. In ordinary circumstances, however, the pressure is usually considerably raised and, according to Meisen, may be as high as 35 to 55 cubic centimetres of water and 6 to 10 cubic centimetres higher if the leg tested is weight-bearing. This is a very considerable pressure when compared with that of a few cubic centimetres in normal veins.

Thus in a varicose venous system the blood has to move heartward against a very considerable pressure and has a very inadequate mechanism to help it on its journey. The net result is stagnation, to a greater or less extent, in the capillaries, venules and veins.

It is this stasis which predisposes to the nutritional changes in the skin and superficial tissues, and these changes lead to atrophy of the skin, eczema, ulceration *et cetera*.

The muscles and ligaments are also affected; patients tire quickly and develop aches and pains in the legs.

Therefore, it is a tremendous advantage to the patient to have these channels sclerosed and to allow the deep vesesls to carry on the venous return unimpeded.

Other reasons for treatment are the unsightly appearance (the main reason in the milder degrees of varicosity) and the possibility of severe hæmorrhage from a ruptured vein.

Treatment.

It is only within recent years that the older methods of treatment, namely, (*a*) masterly

inactivity, combined with the use of elastic stockings and bandages, and (*b*) operative removal of the veins, have been seriously challenged.

Injection Treatment.

Injection treatment was first advocated almost one hundred years ago, but the results were bad, mainly on account of the failure to adopt an aseptic technique, and the method was abandoned. It was the work of the French school, starting about 1915, which placed the method on a firm footing, and these workers cannot be too highly commended for their brilliant research. It was not until 1925, however, that articles began appearing in the British journals and that the method became established in Britain.

Compared with operation, the new method has everything in its favour. Excision was a long, tedious operation, convalescence was often protracted, and recurrence only too frequently followed. The new method is ambulatory, injections may be given without any elaborate apparatus, it is practically painless and does not require an anæsthetic, and each injection takes only a few minutes of the patient's time. In addition, the results are better, and it is safer.

It was the fear of pulmonary embolus that delayed the popular adoption of the technique; but, within the last few years, numbers of surgeons have reported large series of cases without fatality. It is reported that workers in Sicard's clinic have given 300,000 injections with sodium salicylate and 25,000 with quinine without a case of pulmonary embolus. McPheeters and Rice report 53,000 cases with seven deaths, four of which were due to pulmonary embolism (that is, one in 7,500 cases), and Maingot has treated 5,000 patients with only one case of pulmonary infarction, in which recovery occurred. This compares very favourably with 1,444 excisions by Jeannel with four pulmonary emboli and three pyæmias (that is, one in 200), and 4,607 excisions reported by Kilbourne with a death rate of one in 250.

The reason for this practically complete freedom from embolism is the nature of the reaction produced by the injected fluid. The endothelium of the vein is irritated by the solution, becomes congested and hypertrophies. Over this injured endothelium blood fibrin is deposited and adheres firmly. The clot is tough and leathery and adheres over a large area. After twenty-four hours the clot can be separated from the vein wall only by dissection. It rapidly organizes and shrinks to a firm fibrous cord. This is in marked contrast to the ordinary soft *ante mortem* thrombus, lying more or less free in a normal vein, which can easily be detached by a rise in the venous pressure, such as is occasioned by movement. So slight is the attachment that, after the clot has been moved on, it is often very difficult to see any change in the endothelium at all.

Before any actual treatment of the veins is instituted the patient must be carefully examined and any of the before-mentioned exciting factors noted, with a view to treatment. Also it is necessary to remember that all prominent veins are not varicose.

Solutions for Injection.

Solutions which may be used are as follows:

Quinine Hydrochloride.—Quinine hydrochloride (four grammes) and urethane (two grammes) in thirty cubic centimetres of distilled water is probably the most popular sclerosing solution in general use today. It is an excellent scleroser and is painless. Some people, however, have an idiosyncrasy to quinine, and alarming symptoms of cinchonism may be produced. On this account the first injection should always be small, for example, 0·25 to 0·5 cubic centimetre. Also, quite a number of patients complain of nausea and giddiness and feel faint after injection. Maingot puts the proportion down at approximately 10%. Also it is an abortifacient, may cause uterine colic and menorrhagia, and should not be given during menstruation; but these factors are of minor importance.

Lithocaine.—Lithocaine (lithium salicylate 30% and tutocaine 1%) has been used largely by Maingot, under whom I had the privilege to work. In his opinion it is the best solution produced so far, and it has been constantly used in his clinic since 1926. It regularly produces a hard extensive clot and is more reliable in its action than the quinine-urethane solution. It, however, has its disadvantages, the main one being that it must be freshly prepared. It combines with the iron in glass and after a week or two becomes discoloured and deteriorates. It is easy, however, to obtain the requisite amount of powder and, by the addition of sterile distilled water, make the solution oneself. It is in every way more efficient than sodium salicylate, with which it must not be confused. The object of the tutocaine is to mask the sharp stabbing pain that lithium salicylate by itself produces. The usual dose is about four cubic centimetres.

Sodium Chloride and Tutocaine.—Sodium chloride 20% with tutocaine 1% is useless for big veins, but is said to be ideal for small superficial veins. The dose is two to fifteen cubic centimetres.

Sodium Morrhuate.—Sodium morrhuate 5% or 10% is widely used and has many advantages. Large doses may be given, several veins being injected at one sitting. It is painless and it rarely produces an injection ulcer if small amounts be injected subcutaneously. It has several disadvantages, however. The most important is that about 30% of veins apparently satisfactorily sclerosed become recanalized. Another is that it occasionally causes alarming symptoms of collapse, which, though eventually recovered from, are very disturbing to patient and surgeon alike. Urticaria also sometimes follows the injection. On these grounds the use of this solution has been abandoned by many surgeons, and I consider the indications for its use are very limited.

The "Twin Injection".—Quinine-urethane and lithocaine are incompatible, and when mixed

together produce a stringy, glutinous precipitate. This incompatibility is utilized in the twin injection. If two cubic centimetres of quinine-urethane and four cubic centimetres of lithocaine are injected into the same vein, about five centimetres (two inches) apart, they produce a very firm and extensive clot, even when either solution used singly has proved ineffective. Two syringes must be used and as little time as possible must be lost between the injections. Maingot, with the help of an assistant, gives both injections simultaneously, and he has done 1,000 injections without a failure or any untoward symptom. My own experience with this injection, though more limited, has been just as happy, and I have not yet had a failure with it. Only recently I had a case in which the veins failed to react to quinine-urethane or lithocaine separately, but in which a firm clot formed after the twin injection.

Technique.

An ordinary "Record" syringe is quite satisfactory, although I prefer the Jones varix syringe. I use a 16 or 17 hypodermic needle with a medium bevel. If the point is too tapering, it is very easy to transfix the vein. The injection may be given with either a full or an empty vein. In the full vein technique the injection is made with the leg dependent—the patient sits on a raised stool, or stands. In the empty vein technique the patient lies down. This is sometimes useful for big veins, as otherwise the large amount of blood in the vein so dilutes the fluid that the maximum effect is not obtained. This method is made easy if a pneumatic tourniquet is applied with the patient standing and the veins full. The patient then lies down, the needle is inserted into the vein and a little blood is withdrawn. The tourniquet is then released and after the blood has flown out of the vein, the injection is made. Personally, I am quite satisfied with the full vein technique and have never resorted to the empty vein method, but it is worth trying in the event of failure.

The Injection.—The syringe is loaded with the fluid to be injected and then a little water is sucked up, so that there is no sclerosing fluid in the needle itself. This is a prophylactic measure against blockage of the needle. The skin over the vein is dabbed with spirit, the vein is steadied and the needle is inserted into the lumen. A little blood is sucked back into the syringe to make sure that the point is within the lumen, and the injection is slowly made. The injection must not be proceeded with unless the operator is positive the fluid is being injected into the lumen, and the patient is instructed to report any pain after the first prick of the needle. After the injection the leg is raised slightly and the skin puncture compressed with a swab dipped in spirit. When the bleeding (if any) has ceased, the puncture is sealed with a small piece of "Z.O." strapping. If there are no untoward symptoms the patient is allowed to walk away in a few minutes, but is warned not to indulge in excessive muscular exercise.

At the first injection 0·5 cubic centimetre of quinine-urethane solution is given in one of the lowest varicose veins, and at subsequent injections progress is gradually made up the leg, the twin injection being used for the big veins and either solution separately for the smaller ones. The injections are made at weekly intervals. Clotting does not occur immediately, but usually within twenty-four hours the vein is firm and tender, the degree of tenderness varying considerably. Thereafter the vein shrinks until all that is left is a fine firm cord and, perhaps, a line of pigmentation along the course of the vein.

Disadvantages of Injection Treatment.

There are certain disadvantages to injection treatment.

Recurrence.—Recanalization is rare after injection with quinine-urethane or lithocaine (about 2% in Maingot's series), but it is much commoner after sodium morrhuate (about 30%). New varicose veins may form, due to the continued action of the primary pathological processes, especially if the perforating veins are inadequately cut off. Inadequate sclerosing of the higher veins, in which case the recanalization is aided by the weight of the column of blood, is said to be an important factor, and ligation is sometimes performed; but this is not absolutely without risk. Otherwise, recurrences are treated in the same way as the original veins. Kilbourne, comparing recurrence rate statistics, finds 30% after operation and only 6% after injection.

Injection Ulcer.—Injection ulcer is the bugbear of the injection method. If any sclerosing fluid is injected subcutaneously, necrosis of the tissues is liable to follow. Sodium morrhuate is less likely to cause this distressing result than quinine-urethane and lithocaine, which latter two Maingot assesses as equally dangerous. An ulcer is much less likely to follow if the skin and tissues are healthy than if they are thin and atrophic. The first thing complained of is pain while the injection is being given, and perhaps the operator notices a swelling at the site of the injection. In two or three days a dark gangrenous patch appears and this slowly sloughs out, leaving a deep, circular, punched-out ulcer, very like that produced in tertiary syphilis. This usually takes several weeks to heal, but, although inconvenient, is neither painful nor dangerous. If the operator realizes that some of the fluid has been injected subcutaneously, the only thing to do is to inject several cubic centimetres of normal saline solution into and around the area. This frequently dilutes the fluid sufficiently to prevent ulcer formation.

Contraindications to Injection Treatment.

There are certain contraindications to injection treatment.

1. During pregnancy. Raymond Green reports 25 cases injected with quinine-urethane without miscarriage or uterine symptoms, but I consider

treatment during pregnancy inadvisable unless there is some special indication.

2. Advanced cardiac, pulmonary or renal diseases and diabetes are contraindications.

3. Marked cirrhosis of the liver is a contraindication.

4. Active phlebitis. No attempt must be made to sclerose a vein which is the seat of an infection. After the attack has completely subsided, injections may be undertaken if necessary.

5. Whether old deep thrombosis is a contraindication or not is a much debated point. Its presence should be suspected if there is much swelling, œdema, or difference in the colour and temperature of the two legs. The question is: Are the varicose superficial veins compensatory, or are they simply an added embarrassment to a hard-pressed deep circulation? From our knowledge of the blood flow in varicose veins it is difficult to imagine that they can help much in the venous return, and, if we can have arterial collateral circulations, surely we can have venous? Dickson Wright is very definite on the subject and has now treated 56 cases of ulcer associated with old "white leg". In 42 of these, large varicosities were injected, and in the remaining 14, there were no varicose veins present. In addition to these a large number of similar patients suffering from periphlebitis or swelling have been treated by injections, but Wright has never seen any damage in the shape of increased swelling or cyanosis of the limbs result. Turner Warwick agrees that the varicose veins are usually an encumbrance, and suggests the following test. Two elastic bands are placed around the leg, enclosing a segment of vein full of blood and a perforating vein. The patient is made to walk up and down the room, and if the vein empties, the deep circulation, to the next perforating vein at least, is competent. If this test is satisfactory, injection is cautiously carried out.

Maingot, however, is just as definite that it is a contraindication, and he says that if one leg is an inch or two larger than its fellow, injections are contraindicated. My own feeling in the matter is that in these cases injections may be used, but that a degree of caution is advisable.

Varicose Ulcer.

A paper on varicose veins would not be complete if some reference were not made to the commonest complication of that condition, namely, varicose ulcer. Varicose veins are present in about 75% of patients suffering from varicose ulcer, but every case of varix does not necessarily develop an ulcer. What additional factors are necessary for ulcer formation are not definitely known, but the patient's vitality and the occurrence of some degree of trauma, with or without superadded infection, probably play an important part.

However, the main cause is the stagnation of blood, lymph and tissue fluid, which, we have seen, occurs in the varicose leg, and the old methods of treatment failed because this was not realized. All the attention used to be focused on the ulcer itself, and numerous and varied were the methods adopted. All were alike in one respect—they failed to give a permanent cure in all but a small percentage of cases. One humourist stated that the only cure was an income of £400 a year and unlimited rest. It was Dickson Wright's excellent work which focused attention on the final underlying cause, namely, the effect of gravity. He demonstrated that it is the areas subjected to the uniform and constant pressure of the footwear which do not become ulcerated. Thus the treatment to be adopted is the application of some form of constant pressure to the leg, and the ulcer is allowed to look after itself. All through the treatment this fact must be remembered: it is not the ulcer that is being treated, it is the underlying stagnation of venous blood, lymph and tissue fluid.

Details of Treatment.

After the usual examination of the patient the ulcer is simply swabbed with normal saline solution and an elastoplast bandage is applied to the leg. There is no dressing applied over the ulcer. The bandage, which consists of a zinc oxide adhesive dressing applied to a crepe bandage, is first applied to the foot just behind the toes. After encasing the foot, a few turns are made round the ankle, the heel usually being avoided, and then it is continued in the ordinary spiral fashion up the leg, care being taken to overlap each turn of the bandage by half to two-thirds of the width. A good deal of force must be used in applying the bandage, and it must be pulled really tight. Because of its elasticity the bandage moulds itself smoothly to the shape of the leg. The bandage is allowed to remain on for a varying length of time, depending on the conditions present. If the leg is very œdematous, it shrinks rapidly after the application of the first bandage, which then becomes so slack that it is useless after four to seven days and must be reapplied. After the original shrinking has taken place, however, it is allowed to stay on two or three weeks at a time, and as healing progresses it may be left on for even longer periods.

The discharge from the ulcer soaks through the bandage and is washed off the outside daily. Patients must be told to expect the discharge to soak through and must be warned that there is no need to worry. The method of allowing the ulcer to be bathed by its own discharge is similar in principle to that of the Winnett-Orr technique for osteomyelitis.

While this treatment is going on, any varicose veins present must be sclerosed. High veins can be injected with the bandage *in situ*, and one of the lower veins each time the bandage is removed. It is not wise to inject the veins near the ulcer, as severe perivenitis or actual necrosis may follow. When the ulcer is quite healed, however, these veins must be sclerosed if a permanent cure is to be expected. At the first examination of the leg very

few varicose veins may be seen. They are often practically buried by the œdema, and are generally brought to light as this is reduced by treatment.

After even the first bandage is removed, a marked improvement will be noticed in the appearance of the ulcer. It already looks clean and healthy, and the raised callous edges have disappeared. The rate of healing is rapid; Dickson Wright estimates that one square inch is epithelialized each week. It is difficult by ordinary bandaging to exert sufficient pressure on ulcers behind the malleolus, and in these cases a piece of sorbo rubber, slightly bigger than the ulcer, is incorporated between it and the bandage. In large ulcers skin grafting must sometimes be resorted to. Pinch grafts are buried in the granulations, or skin threads are interlaced on its surface, and an elastoplast bandage is applied in the usual way. As Dickson Wright states, "the grafts take with monotonous regularity".

After-Treatment.

When the ulcer is healed and all visible varicose veins have been injected, it is usually necessary to continue supporting the leg for some months by elastoplast, viscopaste, or an elastic stocking, and periodic inspections of the legs should be made to see that adequate care is being taken of it.

Contraindications.

There are only two contraindications to this treatment of varicose ulcers: (*a*) arterial disease, (*b*) diabetes. Constant pressure on the leg of a diabetic is liable to start gangrene.

As I said before, previous deep phlebitis is not a contraindication.

Advantages.

There are three great advantages that this method has over any other:

1. The final results are good, and in experienced hands a cure is certain.

2. The method is ambulatory. The long periods of rest which previously played such an important part in the treatment, are now a thing of the past, and the patient is even encouraged to take exercise and return to his ordinary work.

3. It is cheap. Bandages are fairly expensive (about 4s. 6d.), but not many are needed, and when one thinks of the saving of time lost, the cost seems small.

Disadvantages.

There are certain disadvantages:

1. Failure may be due to incorrect technique, usually in not applying the bandage tightly enough, or incorrect diagnosis. One must not forget syphilitic ulcers are fairly common in this region, and that malignant ulcers sometimes occur.

2. Recurrence sometimes occurs. Wright has had a 3·4% recurrence rate in three years. However, these respond to treatment, and a cure for even a short time, if so easily obtained, is worth while.

3. Pain is often complained of at first, due to the tightness of the bandage; but this soon gives way to a comfort which has not been experienced since the onset of the condition. Sometimes, however, the pain persists, and then it is often due to faulty bandaging, and frequently to an edge of the bandage cutting into the granulation tissue of the ulcer. To avoid this it is advisable to place a strip of elastoplast over the ulcer, and sometimes a longitudinal strip down each side of the leg, before the bandage is applied. Powdered aspirin dusted on the ulcer is a useful analgesic.

4. Blisters sometimes occur. They discharge and cause some degree of pain, but should not stop treatment.

5. Eczema occurs in 5% to 10% of patients, probably due to some idiosyncrasy. It is seldom necessary to stop treatment, but a change of bandages to semiplast or viscopaste may be advisable.

Treatment by Unna's Paste.

In conclusion, some mention must be made of the treatment of varicose ulcers by means of Unna's paste. The ordinary method of applying Unna's paste yields much less satisfactory results than those obtained by means of elastoplast. However, in an attempt to find a cheaper method for use at the Adelaide Hospital, I have started using Unna's paste in conjunction with a crepe bandage. The paste is painted on the leg and a crepe bandage is applied tightly over it. A further layer of paste is applied and the whole covered by a flannel or cotton bandage. In the few cases in which I have so far tried this method, the results have been sufficiently gratifying to encourage me to persist with it.

ACKNOWLEDGEMENTS.

I wish to acknowledge my great indebtedness to the following books and papers, from all of which I have drawn freely:

1. "The Rational Treatment of Varicose Veins and Varicocele", by W. Turner Warwick.
2. A paper on varicose ulcer by Dickson Wright, which appeared in *The British Medical Journal* of September 26, 1931.
3. "The Injection Treatment of Varicose Veins and Hæmorrhoids", by R. H. Maingot.
4. "The Treatment of Varicose Veins by Injection", by V. M. Coppleson.

I am further indebted to Dr. Maingot and Dr. Coppleson for personal communications.

PATHOLOGY IN ITS RELATION TO DIAGNOSIS AND TREATMENT OF CANCER IN THE CANCER CLINIC, ADELAIDE HOSPITAL.[1]

By L. B. BULL, D.V.Sc.,

AND

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IN order that our experience in the treatment of cancer by irradiation may be built on a sound pathological basis, it is advisable that the histology

[1] Read at the Fourth Australian Cancer Conference, Canberra, March, 1933.

of each neoplasm to be treated should be studied and recorded.

In the treatment of cancer by operative surgery, material becomes available for histological study in the ordinary course of events. Unless a small portion of the tumour to be treated by irradiation is removed before treatment is instituted, material does not usually become available.

We believe that material can be removed from any accessible neoplasm for biopsy study without prejudice to the patient. We believe that rough palpation and manipulation of a tumour may assist in the dissemination of neoplastic cells on occasion, but we do not believe that excision of a portion of the growth by a sharp instrument is liable to assist in such dissemination.

Although an experienced clinician will be able to form a sound opinion as to the nature of the majority of neoplasms that are to be seen in a clinic, it must be admitted that the most experienced will sometimes be misled by appearances. This possibility applies more particularly to the clinical differentiation of squamous cell epithelioma from rodent ulcer. As the behaviour of these tumours differs the one from the other, it is advisable in all cases to know the exact nature of the lesion to be treated. Prognosis may depend very largely on the correct classification of the neoplasm.

It must be concluded, therefore, that for the purposes of statistics, for sound controlled experience, for prognosis and for guidance in treatment, all accessible neoplasms should be subjected to biopsy. The fact that the tissue is removed for biopsy study presupposes that treatment will be proceeded with as soon as possible. In actual practice at the Adelaide Hospital the biopsy material is usually removed at the time treatment is commenced.

We do not claim that the histological examination of biopsy material will always reveal the nature of the lesion. In the majority of cases the recent graduate fails to secure a satisfactory piece of tissue. Judgement and experience are necessary in the selection and removal of satisfactory specimens. Removal must be deep enough to show the behaviour of the neoplastic cells. For the most part smaller or shallower portions of tissue will reveal the true nature of a rodent ulcer than is the case in squamous cell epithelioma, this being often associated with much epithelial hyperplasia and hyperkeratosis and requiring a much deeper excision to reveal the infiltration of the tissues by neoplastic cells. Some tumours show more or less extensive areas of necrosis, and care must be exercised to select material as free as possible from necrosis. Larger portions of tissue are usually required for a satisfactory histopathological examination of the sarcomata. Furthermore, it may be difficult for the pathologist to form an opinion on a piece of tissue which may be too small to show a complete picture of the behaviour of the neoplastic cells and the natural variation in morphology and group arrangement. These difficulties place definite limitations on the value of histopathological examinations of biopsy material, and the clinician should never hesitate to question the correctness of a histopathological report if it conflicts with the opinion reached on clinical grounds. Personal consultation of clinician and pathologist is essential under these circumstances.

Methods Used at the Adelaide Hospital.

Biopsy material is placed in saline solution, immediately after removal, in order to wash the blood from the surfaces of the tissue. It is then placed in 10% formalin in physiological saline solution. The following day frozen sections are cut and stained by hæmatoxylin and eosin. Even the smallest pieces removed from rodent ulcers can be easily orientated and successfully handled by this method. The preliminary washing in saline solution assists greatly in the correct orientation of the tissue. In most cases sufficient material is left, after taking a few frozen sections, for paraffin embedding if necessary. With this method a report is available within twenty-four hours, and this early report is valuable in most cases. If an earlier report is required, the tissue in the formalin-saline mixture is placed in a water bath at 50° to 56° C. for half an hour, when fixation is usually complete, and frozen sections can then be made as easily as with material fixed by the longer process. All the routine histopathological diagnoses are made by the use of this method, but material for special study is embedded in paraffin.

The Object of Biopsy Study.

The object of biopsy study is to establish by histological methods the type of neoplasm, and to assess, as far as possible, the degree of malignancy. As the result of experience of many workers over a period of many years, certain tumours arising in certain parts of the body are known to behave in a certain way, according to the age of the patient and other factors. We have learned to associate definite histological appearances with these various tumours. A microscopic examination is not in the nature of a chemical or biological test. The picture revealed has to be interpreted. Without a knowledge of all the facts obtained by clinical investigations, it may not be possible for a pathologist on histological evidence alone to give a complete opinion on the nature of the neoplasm and the prognosis.

For the most part it is possible to determine the type of neoplasm from a histological study of biopsy material. Sometimes, however, appearances may deceive. We may receive, for example, a small piece of tissue from a malignant melanoma, but no pigment may be present in the portion removed. In such circumstances we would probably express the opinion that the tumour is a malignant melanoma *sine* melanin, which would not be the whole truth. Again, we may receive a small portion of a breast tumour which shows the typical histological picture of a scirrhous carcinoma, whereas another section of the same tumour might be far from scirrhous in

character. We believe, therefore, that the finer classification of neoplasms may be somewhat dangerous if biopsy material alone is available.

The pathologist is expected to indicate the degree of malignancy of the tumour under investigation. It is our practice to report the nature of the tumour only, unless, in our opinion, the indications are that the tumour possesses a higher degree of malignancy than is usually found for that class of tumour, or, in the contrary, the malignancy is of low grade. If we report that the tumour is a squamous cell epithelioma, for example, the surgeon understands that the tumour would possess that degree of malignancy which accumulated experience has come to associate with such a tumour, according to the site of origin, age of patient, and so on. Unless the association of the pathologist and clinician is much closer than is usual in this country at least, any attempt to go further than this is liable to be misleading. The histological signs of malignancy measure the potential malignancy of a tumour, but the clinical course is subject to wide variations.

A careful investigation of gross material makes possible a more accurate prediction of the course a given neoplasm is likely to take. As an example we may mention that a tumour of the stomach was submitted to one of us some years ago.

> The histological examination showed a carcinoma of high malignancy. The patient was a young man of twenty-four, and symptoms had been in evidence for a fortnight only. Fortunately for the patient, the tumour had fungated into the lumen of the stomach, and there was only a limited infiltration of the stomach wall at the base. Taking all these facts into consideration, a favourable prognosis was given, or, to be more nearly correct, it was suggested that if the neoplasm did not recur and kill the patient within six months there was likely to be no further cause for anxiety. The patient is alive and well today. Such an opinion could not have been given on the histological examination alone, which showed one of the most rapidly growing tumours we have ever seen.

The term "malignancy" may not always mean the same thing when applied to neoplasms. It probably always means the ability of the neoplasm to destroy the host, but this will not always be in direct relationship with the degree of anaplasia and the capacity for rapid growth, but may in many cases depend upon the anatomical situation. Yet a so-called benign tumour may readily kill if its anatomical situation is favourable to such an end. The capacity of a tumour to give rise to metastatic deposits is closely associated in our minds with the term malignancy. This capacity will depend on ability of the neoplastic cells to infiltrate or permeate the surrounding tissues, an abundance of thin-walled blood vessels and the ability of the cells to grow in other tissues or organs. It would appear that in the early stages of the disease the normal tissues of the body always offer considerable resistance to neoplastic cells, and that a breakdown of these defensive powers will aid local growth and the formation of distant metastasis.

Neoplasms of the skin form a very large proportion of tumours found in a cancer clinic. There has been a desire in many quarters to classify these neoplasms into many groups or varieties. Dr. J. V. Duhig presented a paper at the last Cancer Conference in which he said that the various modes of grading skin cancers had not seemed satisfactory to him. He has our support when he claims that the estimation of the degree of malignancy in any skin cancer is very much a matter of personal choice or prejudice. The microphotographs presented by Dr. Duhig, together with his comments on them, offer a very good example of this. We find, for example, in his Figure III that the departure from normal illustrated is regarded as a malignant change, but we would not regard it as such. Further, in Figure X is illustrated a squamous cell epithelioma which is regarded as less malignant than that illustrated in Figure XI, but from the histological picture alone we would not have formed such an opinion. It is quite evident that pathologists will hold different opinions on the degree of malignancy in many instances, and these differences will become greater with attempts to make fine distinctions and a multiplicity of grades. We hold that the truth can be approached only after a careful consideration of the whole clinical evidence together with the histological picture. While agreeing that there is a great diversity of structure to be found in new growth of the skin, we have not been able to convince ourselves that mixed growths, squamous and basal cell cancers, are common. We have frequently found both squamous cell epitheliomata and rodent ulcers on the face of the same patient, and we have seen squamous cell epitheliomata arise in the site of rodent ulcers that have been treated. We do not hold with the teaching that keratinized epithelial pearls are not to be found in rodent ulcers. It is apparently that type of rodent ulcer in which epithelial pearls are more or less frequent which Dr. Duhig calls a mixed growth. In certain instances rodent ulcers will show these keratinized pearls, which, in our opinion, represent the reproduction by the tumour cells of small hair shafts. We have seen a number of these tumours, and their history and response to treatment do not differ in any way from typical rodent ulcers.

The Growth and Spread of Cancer.

We may return for the moment to the subject of the assessment of malignancy. Professor D. A. Welsh has said that malignancy is to be judged by an estimate of (i) the invasive growth of the tumour cells and (ii) the contact spread or lateral extension of the cancer process. He has become the main exponent in Australia of "contact spread", which is claimed to be the result of the release of a carcinogenetic substance from the cancer cell, which causes adjacent non-cancerous epithelial cells to become neoplastic. He says that he is satisfied that it is the chief means by which untreated cancer spreads to involve a surface epithelium, and it is also evident in glandular cancers.

We are not convinced of the correctness of this hypothesis. On the contrary, we believe that other interpretations of the phenomena are more likely

to be correct. It is in cancers of the skin that this spread by contact is said to be most in evidence. It is not uncommon to find pathologists giving illustrations of malignant transformation at the base of hyperplastic interpapillary processes of the skin, either in rodent ulcer or squamous cell epithelioma. The interpretation of the phenomena observed in these neoplasms is not obvious, hence different opinions are held as to what they represent. It is probable that cancers do not arise from one single cell that becomes neoplastic, but from a group of cells, usually in a very restricted area. It seems evident that the early neoplastic cells have a very hard struggle to establish themselves, and that the death rate is high. Let us postulate the origin of a rodent ulcer in a small group of basal epithelial cells of the skin or a hair follicle. We believe that these cells multiply and start to invade the epidermis and the corium. Invasion along the base of the epidermis, however, appears to be easier than invasion of the relatively dense corium. Many of these cells spreading along the base of the epidermis die off, as cancer cells are very prone to do. Some, however, do remain and are able to establish foci, from which a more successful invasion of the deeper tissues may take place. The primary difficulty, however, appears to be the invasion of the deeper tissues. Gradually the resistance is broken down, but it may be months or years before the tumour is well established to the naked eye.

It is agreed that there is usually some preliminary hyperplastic change in the epithelium before any neoplastic change develops. After the malignant change has occurred, more hyperplasia of the non-cancerous cells occurs. The cancer cells have, however, made a preliminary permeation, and many neoplastic cell groups remain to carry on invasion. Thus the appearances may suggest that a neoplastic change can be traced in adjacent non-cancerous groups of cells, but we believe this interpretation to be wrong and that the explanation is to be found in early permeation with death and disappearance of many of the connecting strands of neoplastic cells. The capacity of neoplastic cells to make widespread and insidious permeation is well illustrated in cancers of the breast and in cancers of the stomach in the form often called *linitis plastica*. It is most difficult to interpret the microscopic appearances in many neoplastic processes, particularly when the cancer cell is invading the tissue from which it has originated, such as occurs with a squamous cell epithelioma or a rodent ulcer. The so-called neoplastic change at the base of a hyperplastic epithelial process is, in our opinion, more often an invasion of the process from below. This cannot be proved, for the cells are too similar in appearances, but a similar picture may be found when a cancer cell of different origin invades the skin or a mucous surface. Figure I illustrates such an invasion of the nasal mucosa by a squamous cell epithelioma. In this case the cancer cells did not originate in the surface epithelium, but grew through the antrum.

It may be argued that the absence of any evidence of contact spread in metastatic deposits is due to the carcinogenetic substance being highly specific and capable of acting only on cells of similar embryonic origin to the cancer cells. This argument cannot be applied, however, when a cancer of the gastro-intestinal canal gives rise to deposits in the liver, yet we do not see any evidence of contact spread in these circumstances.

FIGURE I.

Invasion of the nasal mucosa by a squamous cell epithelioma from the antrum. The epithelial processes extending to the deeper tissues are not hypertrophic processes showing early neoplastic changes, an explanation usually advanced for similar appearances in skin tumours, but are formed entirely of neoplastic cells. No squamous epithelium exists normally in this situation, therefore these processes must have developed from the tumour cells or by metaplasia, the former being the more probable.

There is not sufficient time or space to develop this argument further here. The matter is, however, of some practical importance and not merely of academic interest.

After a consideration of the subject of malignancy, what the term means and how it can be recognized and assessed, we find that it would be a difficult matter to get all pathologists to agree. In our opinion it is not an easy matter to assess malignancy with any degree of certainty by microscopic examination alone. We therefore favour the use of restraint by pathologists in reporting upon this aspect, particularly when biopsy material alone is available for study.

Some Observations on the Result of Treatment by Radium and X Rays.

No attempt will be made in this paper to summarize the effects of treatment, this already having been done for all the clinics by Dr. M. J. Holmes. There are certain observations which, however, may be made here.

We should like to draw attention to the occurrence of squamous cell epithelioma in or near the site of a rodent ulcer which has been treated.

The first case of this kind was in a man of fifty-four years who had a rodent ulcer of the lip. The lesion consisted of an ulcer with craggy base and heaped up edges, occupying the central third of the lower lip at the muco-cutaneous junction, and measuring 3·0 by 1·5 centimetres. The lesion was superficial. A small ulcer was noticed on the lip five months previously. It was painless, did not bleed, and crusts separated from it from time to time. A portion of the tumour was removed for microscopic examination, and the histological picture was typical of a rodent ulcer (see Figure II). Treatment consisted in needling with monel filtered needles, 0·5 millimetre in thickness, and a dose of 1,176 milligramme-hours was given. He was seen two months later, when reaction was still evident, with improvement in the ulcer. Three months later still, he showed an increase in the size of the ulcer, with a loosely attached crust. There was necrosis along some of the needle tracks. A month later (that is, seven months after the first treatment), when a still further increase in the size of the ulcer was evident, a

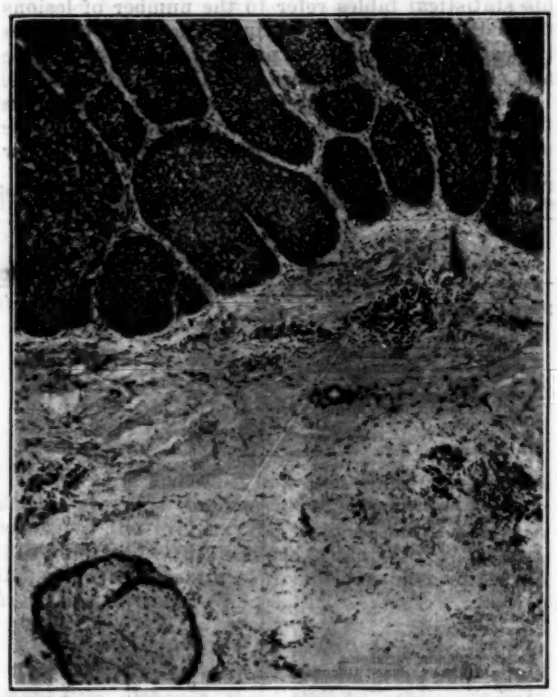

FIGURE II.
Section of tumour of the lip, Case I, showing a typical rodent ulcer.

second treatment with platinum needles, 0·5 millimetre in thickness, in a dose of 1,152 milligramme-hours, was given. A biopsy was also made at this time and a frank squamous cell epithelioma was found, no rodent ulcer cells being found in any part of the section (see Figure III). His after history is good.

A second case, with a somewhat similar result, was that of a man, aged eighty-four years. He showed a large tumour, four by three centimetres, involving the bridge of the nose, extending to the supraorbital ridges and showing three ulcerated areas. Nine years previously he had noticed a lump on the right *ala nasi*, which ulcerated

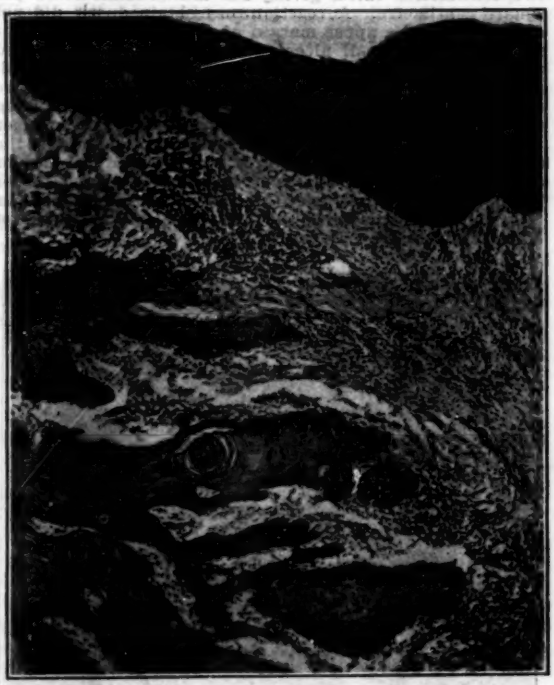

FIGURE III.
Section of tumour of the lip, Case I, showing a typical squamous cell epithelioma. Section taken seven months after treatment began.

and travelled across the nose, but which healed within three years of its onset. A small swelling on the bridge of the nose was noticed five months before his admission to hospital and had developed into the lesion described. A section removed for microscopic examination showed a rodent ulcer. Treatment consisted in embedding monel needles, 0·5 millimetre thick, and a dose of 1,500 milligramme-hours was given. One month later the ulcers were more extensive, but reaction was evident. A slight decrease in the size of the ulcers followed, but three months after the commencement of treatment the ulcers were spreading, with some displacement of the left eye. The tumour continued to increase in size and hæmorrhage had to be controlled by giving a dose of deep X rays. He died from pneumonia four and a half months after first being seen, and a microscopic examination of the tumour revealed a squamous cell epithelioma. No metastatic deposits had formed.

The third case was that of a woman of sixty years, who showed a small raised tumour, 1·5 centimetres in diameter, below and outside the right outer canthus. She had noticed a small pimple in this region two years previously, which had slowly increased in size. A portion of the lesion was examined microscopically and gave a picture of a rodent ulcer. Treatment consisted of the application of a radium plate (half strength, three by three centimetres for three and one-third hours), the application being repeated after two months. A good reaction was obtained after both applications. One month after the second application she had a fixed scar with surrounding infiltration and attachment to underlying bone. Four months after the second treatment a radium pack (two

centimetres thick, giving 210 milligramme-hours per square centimetre) was applied. The application was inefficient and only a slight reaction was obtained. Eight months after this a small ulcer appeared in the centre of the scar, sections of which showed a rodent ulcer. This was treated a month later by the application of a pack two centimetres thick, giving 366 milligramme-hours per square centimetre. A reaction was obtained, but did not extend above the upper margin of the scar. Improvement resulted, but a small ulcer remained, which still showed rodent ulcer cells. Radium needles were then applied, two years after admission, platinum needles, 0·5 millimetre thick, giving a dose of 1,680 milligramme-hours, being used. A good reaction was obtained, a persistent ulcer with a bony floor being left. A small hard nodule at the upper margin of the reacting area was observed five months after the needling. This was removed two weeks later by endothermy. Sections of this showed an early squamous cell epithelioma arising at the base of an area of hyperkeratosis (see Figure IV).

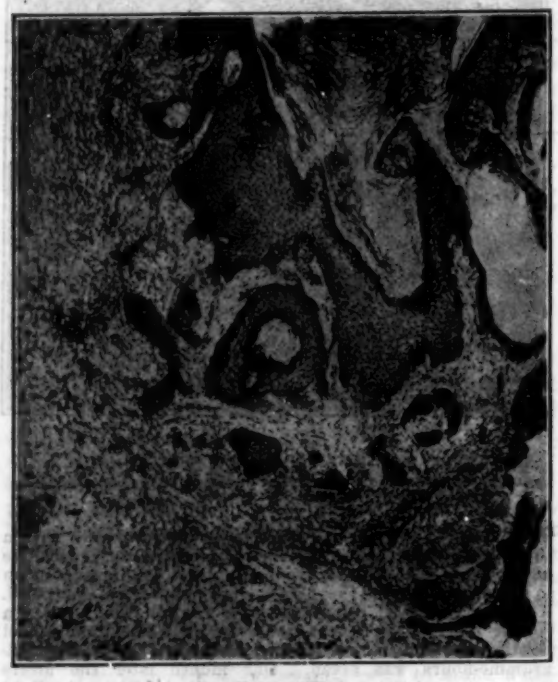

FIGURE IV.
Section of a small nodule occurring at the margon of the reaction area, Case III. The picture is that of the early development of a squamous cell epithelioma at the base of an area of hyperkeratosis.

It is a difficult matter to decide whether treatment played any part in the development of the squamous cell epitheliomata in these cases. The first case offers the most clear-cut observations and the results suggest that the irradiation was effective in destroying the rodent ulcer cells, but that it may have caused some change either in some of these cells or in the adjacent epithelial cells which led to the development of a squamous cell epithelioma. Certainly there was nothing in the microscopic section to suggest the presence of a mixture of rodent ulcer and epithelioma. The dose of irradiation was larger than that usually given for the treatment of epitheliomata of the lip of the same size, but the filtration was less than that now employed.

As no definite conclusions can be reached after a consideration of these cases, they are simply put on record as possibly indicating the carcinogenic effect of irradiation in a small proportion of cases under special circumstances. In support of this tentative conclusion and illustrating the stimulating effect of irradiation at times, we may mention another case.

A woman of sixty-two years showed a firm raised tumour, two centimetres in diameter, with a little surrounding induration, at the centre of the dorsum of the left hand. Sections showed a squamous cell epithelioma. She was given the customary dose with a radium plate, and six weeks later the tumour was approximately three times larger and fungating. A section showed much loose vascular stroma associated with the tumour cell masses. The tumour was removed by endothermy and the base was treated with buried needles. The lesion healed, but fourteen months after the first treatment a broken-down epitrochlear lymph gland was found and amputation of the arm was performed.

It would appear that the first treatment by irradiation in this case caused a stimulation of the tumour.

The Relative Frequency of Types of Neoplasms in the Clinic.

The figures supplied to the Commonwealth Department of Health and used by Dr. Holmes in his statistical tables refer to the number of lesions treated. In the accompanying table we give figures indicating the number of patients seen at the clinic and showing various types of malignant neoplasms. For this purpose individual patients have been taken. Neither the number of lesions nor the number of treatments has been considered. A few

Table showing Relative Frequency of Types of Neoplasms in Patients Treated at Clinic during the period July 1, 1929, to December 31, 1932.

Disease.	Number Treated.	Percentage of Whole.
Malignant epithelial tumours—		
Skin—		
Rodent ulcer	408	30·2
Squamous cell epithelioma	152	11·2
Sweat gland adeno-carcinoma	1	—
Lip	141	10·4
Tongue and mouth	73	5·4
Pharynx, larynx and antrum	27	2·0
Œsophagus	18	1·3
Stomach	31	2·2
Large bowel	8	0·6
Rectum	15	1·1
Breast	151	11·2
Uterus—		
Cervix, squamous cell	60	4·4
Cervix, adeno-carcinoma	4	0·3
Body, adeno-carcinoma	14	1·0
Chorion-epithelioma	1	—
Prostate	31	2·2
Bladder	20	1·4
Penis	4	0·3
Vulva	7	0·5
Testis	9	0·7
Ovary	25	1·8
Thyreoid	5	0·3
Salivary glands, carcinoma	9	0·7
Salivary glands, mixed tumours	8	0·6
Brain	9	0·7
Lung	4	0·3
Branchiogenic	14	1·0
Pancreas	4	0·3
Various	14	1·0
Sarcoma—		
Skin, subcutaneous *et cetera*	33	2·4
Bone (Myeloid 3, Ewing's 1)	15	1·1
Lympho-sarcoma	12	0·8
Retro-peritoneal	1	—
Melanoma of skin—		
Pigmented	15	1·1
Non-pigmented	17	1·2
Endothelial tumours	15	1·1

of the patients actually received no treatment. When considering cases only, a slight correction has to be made on account of the fact that a few patients, actually 24, or 3·5% of the whole, had both rodent ulcers and epitheliomata. Strictly speaking, therefore, only 678 patients had cancer of the skin and lip, instead of 702 as shown in the table. This has been corrected in estimating the percentage frequency in the 1,351 patients under consideration.

It is possible that well defined differences may exist in the frequency of various types of neoplasms found in different parts of Australia, as has been found in different parts of the world. Unfortunately the figures given will not represent the frequency of these neoplasms in the general community. These might be obtained from the general statistics of the hospitals, but they deal only with admissions and are, therefore, of little use for the purpose. Dr. Duhig has found that nearly 25% of patients treated for cancer in the Brisbane Hospital were suffering from cancer of the skin, either rodent ulcer or squamous cell epithelioma. We have found that, in the cancer clinic at the Adelaide Hospital, 30·1% of the patients treated for cancer had cancer of the skin (including the lip).

Apart from the 1,351 patients treated for malignant new growths, as shown in the table, 209 patients were treated for other conditions, including keratosis and papillomata 165, angioma 8, endometrioma 4, and Hodgkin's disease 22.

Acknowledgement.

We are indebted to Mr. G. C. McLennan for preparing the photomicrographs.

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DIABETES AND EYE DISEASE.[1]

By G. H. Hogg, M.D.,
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DIABETES is a disease in which almost every part of the eye may become affected, so the diabetic lesions of the eye may be most varied. Leber in 1875 published a full account of diabetic eye lesions, and since then many contributions have appeared from time to time.

The Eyelids.

The eyelids may be affected from styes and severe blepharitis, although these may be only accidental complications. I have had only two cases of bad styes occurring in diabetic patients.

The Conjunctiva.

Conjunctivitis may occur and is sometimes monocular. It is, however, a rare condition. I reported a case some years ago in THE MEDICAL JOURNAL OF AUSTRALIA and have seen only three cases in forty years. It yields readily to the treatment of the causal disease, local remedies not being of much value. Koenig, from statistics drawn up in Vichy, records eight cases among 500 diabetics, 60 of whom had eye complications.

The Cornea.

Keratitis is said to occur. Koenig recorded one case of ulcerous keratitis, Galezowski 3% in 144 cases, Lagrange 8% in 52 cases, Schlink 4% in 21 cases, Kako none in 280 cases, Schmidt Rimpler none in 150 cases. I have never seen a case of keratitis *per se*, although I have met with a corneal ulcer in a diabetic due to an injury.

A diabetic neuritis of the fifth nerve may cause a *herpes ophthalmicus* with involvement of the cornea, and I have seen one such case.

The Iris.

That diabetes is a cause of iritis and iridocyclitis is undoubted, and to Leber the discovery of this complication is due. Many years ago I heard the late Jonathan Hutchinson state that in his experience iritis associated with glycosuria almost always occurred in patients who were the subjects of gout. In Tasmania gout is very rare, but I have seen a number of cases of iritis, usually mild in degree, although some were severe attacks.

One occurred in an old woman. The last of several attacks for which I attended her, was of most malignant type and was accompanied by a large hæmorrhage into the anterior chamber.

If one systematically examines the eyes of diabetics, one may from time to time find old synechiæ, the remains of exudates *et cetera*, although the patients can give no history of eye trouble. Of course, in some of these cases the iritis may have been due to other causes. Treacher Collins, Becker and Deutschmann have noted degenerative changes in the pigment layer of the iris. Koenig recorded two cases of iritis among 60 cases of diabetic eye complications. Pure diabetic iritis is said by Arruga to respond very poorly to atropine, particularly if insulin is being used, but to react well to "Glaucosan".

I have noticed in at least half a dozen cases a rigidity and relative immobility of the pupil. It is impossible to effect anything but the slightest dilatation of the pupil with any mydriatic, even solid atropine, and in some of these cases I have tried combinations of atropine, cocaine and adrenaline without success. Three of these patients had retinitis of the punctate hæmorrhagic type. Needless to say, in none of these cases was there trace of old iritic adhesions to explain the failure of dilatation.

I have seen no description of this condition in any of the literature of the subject. My friend, Dr. George Mackay, of Edinburgh, to whom I described the condition, said that he had never met with it, and brought the matter before the notice of Mr. Treacher Collins, who was good enough to write that:

[1] Read at a meeting of the Tasmanian Branch of the British Medical Association on January 17, 1933.

The dilator muscle fibres of the iris are derived and connected with the anterior of the two layers of pigment epithelium on the back of the iris. Hence, if in diabetics the pigment epithelium of the iris is degenerate, the dilator muscle fibres are very likely to be affected and rigidity of the pupil may result.

Paralysis of the oculomotor nerves has been reported from time to time.

According to Dr. James Collier, paralysis of the sixth nerve on one side is most frequent, then paralysis of the third nerve, then bilateral paralysis of these nerves in the same order, then combined paralysis of the third and fourth nerves on one side and of the third and opposite sixth nerve. Isolated paralysis of the fourth nerve he has never seen. Personally, I have seen paralysis of the sixth nerve only, but not of any of the other oculomotor nerves. Dr. Collier believes that the pathology is a matter of hæmorrhage into the nerve trunks, or it may be a lesion of the central nervous system of the vascular origin. Knies believed it to be a peripheral neuritis. Loss of accommodation was noticed by von Graefe to occur in diabetics and apparently may occur early in the disease. In my experience of diabetes I have noticed it in only comparatively few cases.

On the other hand, refraction changes have been seen by me from time to time, and in some cases have been most marked. Affleck Greeves, in a recent communication, refers to the rarity of this phenomenon. I have seen it in a fair number of cases, so that in ordering glasses for diabetics I have warned them of the possibility of this transitory change.

Anderson, out of 82 cases of eye complications, found transitory refraction errors present in 12, refraction errors being the third in order of frequency. He believes, however, that next to retinal disease, the refraction changes are the second in frequency. His figures are as follows:

42 cases, retinal affections	= 52·4%
13 cases, diabetic cataract	= 16·0%
12 cases, transitory refracion errors	= 14·6%
7 cases, vitreous opacities	= 8·5%
1 case, muscle paresis	= 1·2%
1 case, iritis	= 1·2%
1 case, optic nerve atrophy	= 1·2%

The change which may be of the nature of hypermetropia or myopia, is transitory and varying in degree. I have found a variation of one to two diopters in a week or two, and in one patient there was a change of from 1·0 diopter of hypermetropia to 3·0 diopters of myopia. Myopia has, however, in my experience been the most frequent change, and in another of my cases in an eye which was emmetropic a myopia of 7·0 diopters eventually developed in one eye and of 4·50 diopters in the other.

It has been stated that transitory hypermetropia never develops in untreated cases, and appears only at the moment the patient undergoes treatment, and that transitory myopia occurs in untreated diabetes. Duke Elder believes that a transitory increase of refraction develops when the blood sugar is markedly increased, and that a transitory diminution of refraction occurs when the glycæmia falls markedly as a result of treatment. He attributes these modifications to osmotic troubles in the lens causing changes in the index of refraction. This is, of course, only an hypothesis.

An observation of Gransbrom is as follows:

Cases of undoubted diminution of refraction	25
Cases of "doubtful" diminution of refraction	11
Cases of "negative" diminution of refraction	9
Total	45

In other words, he noted 55% undoubted diminution of refraction. Of the 45 patients, 12 had been treated by insulin; eight of them had diminution of refraction. Among the remaining untreated patients, 33 in number, 17 had diminution of refraction. This complication is thus seen to be frequent, even in patients untreated by insulin, and medication need not be considered as having any bearing on the frequency. Personally, I have not been able to satisfy myself that treatment had much influence on the development of the phenomenon.

Glaucoma.

Nine cases of *glaucoma simplex* have been found in diabetics by Dr. Shephardson and Dr. Crawford. I have never seen a case of glaucoma in a diabetic patient, and I should imagine its presence is rather in the way of a coincidence. Bistis believes that the administration of insulin may raise intraocular tension and advises that the intraocular tension should be estimated before its use.

Lens.

For many years it has been known that cataract is met with in diabetic patients, so that it has gradually become an accepted fact that cataract is one of the ocular complications of that disease, the glycosuria being thought to be the cause. Most authors in the past agreed that cataract formed the largest proportion of all these complications. Von Graefe believed that lens opacities were to be found in 25% of all diabetic cases. Koenig found only 2%; Williamson, 8%; others, 3%, 4%, 5% and 6%. Shephardson and Crawford diagnosed with the ophthalmoscope lenticular opacities in 54% of their cases. The following table of Groenouw shows the percentage of complication of five different investigators.

My own experience has been that cataract is by no means the commonest complication of diabetes, retinitis being of more frequent occurrence, and in this respect I have the support of Anderson. In 292 diabetic patients in the Copenhagen hospitals he found 82 eye complications, made up as follows:

Retinal affections	42 = 52·4%
Diabetic cataract	13 = 16·0%
Transit refraction errors	12 = 14·6%
Opacities in *corpus vitreum*	7 = 8·5%
Amblyopia centralis	4 = 4·9%
Muscle paresis	1 = 1·2%
Iritis	1 = 1·2%
Atrophy of the optic nerve	1 = 1·2%

Although for many years it was assumed that diabetes was the cause of these cataracts it is

Condition.	Galezowski (1883). 144 Cases.	Lagrange (1887). 52 Cases.	Schilinck (1909). 21 Cases.	Kako (1903). 280 Cases.	Schmidt Rimpler. 150 Diabetics.
	%	%	%	%	%
Keratitis	3	8	44		
Iritis	5	6	12	1·1	5
Choroiditis	3		4		
Cataract	31	25	52	30	45
Retinal diseases	21	36·5	8	23·5	23
Amblyopia without visible pathological fundus changes	27	4	4	6·1	10
Affections of optic nerve		6	4	3·9	25
Refraction and accommodation changes		2	4	3·6	5
Opacities in *corpus vitreum*				1·4	3
Abscesses in eyelid and orbits		6			
Pareses of ocular muscles	7	2			7

doubtful whether, at any rate in older diabetics, the disease plays any, or at least any important part in the causation. Of course, in the diabetes of young people a cataract of rapid development is sometimes met with which is undoubtedly due to the disease, and probably, as Foster-Moore says, the term "diabetic cataract" should be restricted to this variety. As for the diabetes of older people, the cataract which occurs corresponds in incidence, frequency and general character to the cataract of middle and advanced life and is due largely to the same causes. Diabetes, by its effect on the general health, predisposes to weakness and senility and so helps towards the development of lens changes.

The following table of Anderson is most interesting, showing, as it does, that the frequency of cataract in diabetes in the different age groups after forty approximates closely to that of the non-diabetics.

Age Category.	Percentage of Frequency of Cataract.	
	Non Diabetics.	Diabetics.
41–50	38·2	38·8
51–60	61·5	72·2
61–70	85·4	81·2
71–80	92·9	88·2

As regards the causation of cataract we are quite in the dark. All we can say is, in the words of Parsons, that: "It is a disorder of the nutrition of the lens due to some deleterious agent circulating in the blood stream." The old theory that it was due to the withdrawal of water from the lens in consequence of the presence of sugar in the aqueous is exploded. Dor in fact believed that it was due to hydration or, more properly speaking, hydrolysis. It has been shown that at least 5% of sugar is necessary to produce lens opacity, while Burdon-Cooper estimated the sugar in the aqueous to be not more than 0·45%. He (Burdon-Cooper) believes that the acids and ferments, whatever they may be, which are secreted by the ciliary body into the aqueous are responsible for hydrolytic changes in the lens. He has found in some diabetic cataracts an excess of cholesterin instead of tyrosin, which he had discovered some years ago in the lens.

Optic Neuritis.

I have never seen a case of optic neuritis in a diabetic.

Auto-Toxæmic Amblyopia.

Auto-toxæmic amblyopia (Fuchs) occurs in diabetes, being in the nature of a retro-bulbar neuritis, and several ophthalmologists report undoubted cases. In the majority of the few cases which I have seen I have been unable to exclude the tobacco factor. Apparently the presence of diabetes makes the patient more susceptible to nicotine poisoning.

Disease of the Retina.

Disease of the retina is in my experience the most frequent eye complication in diabetes and usually occurs in people of fifty years or over. I have never met with it in young diabetics, but my experience of youthful diabetics has been small. The youngest patient of Nettleship was thirty-five and of Foster-Moore thirty-nine years of age.

Anderson's findings confirm my experience. Thus of eighty-two eye complications he found that forty-two were due to retinal disease, and thirty-four of these were in people of over fifty years of age. Contrary to the views of some authorities, I have come to regard some kinds of retinal change as pathognomonic of diabetes, and on more than one occasion in the out-patient department, without claiming any special skill, I have diagnosed the condition as diabetic and sent the patient to have the urine tested.

Garrod seems doubtful about retinitis being due to diabetes and states that most of his patients who developed retinitis with diabetes have shown evidence of renal disease likewise. He suggests that cardio-vascular changes and high blood pressure may be the cause. I have frequently seen diabetic retinitis in patients with a normal blood pressure, although, as Dr. Leyton has pointed out, we cannot conclude that because the blood pressure is not raised the vessels of the eye may not have been profoundly changed, perhaps by a toxin.

Shephardson and Crawford, although agreeing that retinitis is the most frequent ocular disease associated with diabetes, state that it is absent in uncomplicated diabetes, and when present is usually accompanied by generalized arteriosclerosis or renal

disease. They state that the retinitis seen with diabetes is primarily the result of cardio-vascular and renal disease modified somewhat by the presence of diabetes. In this they are, I think, entirely wrong. One wonders whether these writers are forgetting what Schobl pointed out long ago, that there may be cases of: (i) diabetic albuminuric retinitis in patients whose urine contains sugar and albumin, in whom ophthalmoscopic examination reveals a picture of retinal changes due partly to diabetic and partly to albuminuric retinitis; (ii) albuminuric retinitis in the eyes of diabetics in whom there is a nephritic process.

It is sometimes forgotten, too, that, as Garrod says, diabetes is no single disease and that cases classed under that heading have often nothing in common beyond the tendency to excretion of sugar in the urine.

Cammidge divides diabetes for practical purposes into two main types: (i) The anapothectic or alimentary variety, in which there is difficulty in storing carbohydrates, (ii) achriatic or true diabetes, in which there is defective utilization as well as defective storage.

As a rule the former occurs in elderly people, and it is with this form that retinitis is usually associated.

1. The most typical retinal condition in my experience is the presence of multiple punctate hæmorrhages, small dark red spots of blood about the size of a pin's head scattered over the fundus. These are at times few in number and at the periphery, consequently they may be missed. They may be present without other change. The hæmorrhages are seldom of the flame shape seen in albuminuric retinitis.

2. White patches and spots may be present, often numerous; they may be rounded, oval or of irregular outline, but the edges are very clearly defined and sharply cut; they may be scattered, but sometimes are grouped irregularly near the macula. They do not show any tendency to assume the typical stellate appearance of albuminuric retinitis, although in a few cases I have seen an imperfect half-stellate arrangement. Small punctate hæmorrhages may be present in addition.

3. The optic disk shows no sign of inflammation and the margins are clearly defined.

It has been stated by Gallus that in diabetics cataract and retinitis could not be met with simultaneously. This is entirely wrong. I have seen cataract, retinitis, vitreous opacities and changing refraction all in the same patient, and more than once after removing a cataract in a diabetic I have, on examination of the fundus a fortnight later, found an old standing hæmorrhagic retinitis.

The prognosis of retinitis is good as far as life, but bad as far as vision is concerned. Most of my cases of retinitis have belonged apparently to the anapothectic group. Treatment with or without insulin has resulted in disappearance of sugar from the urine, but there has seldom, if ever, been any improvement in the retinal condition.

Dr. Cammidge believes that the cause of the retinal hæmorrhages in diabetes is a calcium deficiency of the blood causing diminished coagulability, and he advises the administration of calcium lactate to prevent such hæmorrhages. But Dr. Lawrence doubts the accuracy of this, as in ten cases of diabetic retinitis he found a normal blood calcium. I may say that I have given calcium to some of my diabetic patients with retinal hæmorrhages without any benefit, as far as I could distinguish. It might, however, be advisable in diabetics to administer calcium as a safeguard against retinal hæmorrhages, especially if the blood calcium is deficient.

Of *lipæmia retinalis* I have had no experience in my own practice.

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Reports of Cases.

TETANUS.

By DONALD D. COUTTS, D.S.O., M.B., B.S.,
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W.B., A MALE, aged twenty-three years, was admitted to the Williamstown, Footscray and District Hospital on March 5, 1933, suffering from tetanus. For four weeks prior to admission he had been working on a crusher with a road repairing gang. The hygienic conditions of the camp and the drinking water were not good. Horses were stabled in the camp.

I first saw him on the evening of March 5, 1933. He then complained of pain in the upper part of the abdomen, and stiffness and pain in the neck and back. On the day prior to admission he had difficulty in walking, owing to stiffness and spasms of the muscles of the legs.

His skin was very dirty, with numerous marks of flea bites and scratches on the abdomen and back. No wound could be found on the body, nor in the mouth. There was no history of a cut, sore or bruise. The tongue was

thickly coated, the teeth were very dirty, and the breath was foul. The temperature was 36·7° C. (98° F.), the pulse rate was 100 and the respiration rate 20 per minute. The muscles of the neck were stiff and would go into a tonic spasm on any attempt at movement. The abdominal muscles were rigid. When he attempted to walk the muscles of the legs went into a tonic spasm. *Risus sardonicus* was present. The mouth could be opened. The knee jerks were very active with clonus; ankle clonus was pronounced; the plantar reflex was flexor.

He was given 3,000 units of tetanus antiserum immediately.

On March 6, 1933, there were pronounced head retraction and *risus sardonicus*. The muscles of the legs, back and abdomen were very rigid. Six thousand units of tetanus antiserum were given intravenously. Morphine was given hypodermically in a dose of 0·016 gramme (one-quarter grain), and chloral and potassium bromide were given by mouth.

On March 7, 1933, the condition was much worse. Opisthotonus was very marked, and spasms were frequent. The patient was able to open the mouth, but the tongue was being bitten during the spasms. Lumbar puncture was performed under ether anæsthesia. The cerebro-spinal fluid was under pressure; it was clear, and there was no increase in cells. Three thousand units of tetanus antiserum were given intrathecally, and the foot of the bed was raised.

On March 8, 1933, the condition was worse; the patient was very delirious, and opisthotonus was severe; it was impossible to flex the legs or back except under deep anæsthesia. There were severe and frequent spasms of the whole body. The tongue was badly bitten; but the jaw could still be opened. Swabs taken from the mouth and throat contained no tetanus bacilli. Under general anæsthesia ten cubic centimetres of cerebro-spinal fluid were removed and 40,000 units of tetanus antiserum were given intrathecally; 60,000 units were also given intravenously.

The condition was unaltered on March 9, 1933. There was an urticarial rash on the abdomen and back. Under general anæsthesia 40,000 units of tetanus antiserum were given intrathecally, and 60,000 units intravenously.

Next day the spasms had become less frequent. There was still marked opisthotonus. The respirations were shallow. The temperature was 37·9° C. (100·3° F.), the pulse rate was 104 and the respiration rate 22 per minute. Tetanus antiserum was given as on the previous day.

On March 11, 1933, the condition was improved. The spasms were less frequent. The doses of tetanus antiserum were repeated. General anæsthesia was still necessary.

By the next day the condition had improved greatly, and the patient was sleeping well. The muscles were less rigid, and the patient was able to move his arms and bend his back. Spasms were less frequent and less severe.

On March 14, 1933, a profuse urticarial rash developed on the abdomen and back. Forty thousand units of tetanus antiserum were given intravenously.

On March 15, 1933, the rash commenced to fade. Sixty thousand units of tetanus antiserum were given intravenously.

On March 16, 1933, the patient was able to sit up and to move his arms and legs. There was still some rigidity of the abdominal and neck muscles, but there were no severe spasms. The patient had an irritating cough, and expectorated thick, foul sputum. No tetanus organisms were found in the sputum.

Risus sardonicus was still marked on March 25, 1933. The knee jerks were very active. The patient was convalescent.

The points of interest about the case are as follows:

1. The absence of any wound or focus of infection. It is probable that infection took place in the flea bites or scratches. No septic wound could be found.

2. Trismus was never a very marked feature. The patient was able to take food by the mouth throughout the illness.

3. The effect of large injections of tetanus antiserum intrathecally and intravenously on the course of the disease is also very strikingly shown. Only small supplies of tetanus antiserum were available during the first three days of the illness, and during this time the patient's condition was obviously becoming worse. The first large dose of tetanus antiserum was given on the fourth day. This was repeated on the fifth day. On the sixth day there were signs of improvement. In all, over half a million international units of tetanus antiserum were given during the course of the illness: 3,000 units subcutaneously, 346,000 units intravenously, and 166,000 units intrathecally.

No other treatment, with the exception of the administration of chloral and potassium bromide, and an occasional injection of 0·016 gramme (one-quarter grain) of morphine, was employed.

Reviews.

OTO-RHINO-LARYNGOLOGY.

THE popularity of the book, "Diseases of the Nose, Throat and Ear for Practitioners and Students", edited by A. Logan Turner, cannot be questioned. There have been, during the last five years, two reprints of the second edition and now a third edition has appeared.[1] The book, originally based on the small volume by the late Major W. G. Porter, D.S.O., M.B., F.R.C.S.E., is dedicated to his memory. It "is produced as the joint work of those engaged in the teaching and practice of the specialty in the Edinburgh Medical School". Each section has been carefully revised by the author, certain portions of the text have been rewritten and some new matter has been added. Care has been taken not to increase unduly the size of the volume.

The sections on peroral endoscopy and Ménière's symptom complex have been rewritten and the main addition is the description of the physiology of the otolith apparatus and its relation to clinical investigation. Twenty-three new illustrations have been inserted.

We had pleasure in reviewing the second edition in THE MEDICAL JOURNAL OF AUSTRALIA of April 14, 1928, and we recommended it as being probably the best book on the subject for its size in the English language. We remarked at the time that some of the chapters were so full as to suggest that eventually the book might appear as a comprehensive standard text book. In some phases of the present edition, this standard has been approached and we still hope that in the future it will take its place and fill the requirements which oto-rhino-laryngologists feel have been neglected by British authors.

The book, coming as it does from the Edinburgh School, presents certain characteristics which are more or less typical of that school of oto-rhino-laryngology. Too much emphasis has been made of the operation of inferior turbinectomy. Surgeons in Australia are inclined to reserve interference with this structure for rare occasions. Likewise we again criticize the describing in detail of West's operation for dacryocystitis. We hold that the combined external and internal operation is more satisfactory and more surgical, and it has not received any notice. The use of the flap of mucosa from the outer wall of the nose to cover the floor of the antral cavity after performing the radical antral operation, we consider is not to be recommended. It is pleasing to see that the question of tonsils and adenoids is dealt with in two sections, but it could be emphasized by making a definite statement that the tonsil and adenoid operations should not be considered as one. As a routine, tonsillectomy by means of the guillotine is preferred to dissection, in Edinburgh. Sluder's operation for exenteration of the

[1] "Diseases of the Nose, Throat and Ear for Practitioners and Students", edited by A. L. Turner, M.D., LL.D., F.R.C.S.E., with the collaboration of J. S. Fraser, M.B., F.R.C.S.E., *et alii*; Third Edition; 1932. Bristol: John Wright and Sons, Limited. Demy 8vo., pp. 491, with 250 illustrations in the text and 19 plates. Price: 20s. net.

ethmoidal labyrinth has been described, accompanied by numerous illustrations. This method of dealing with ethmoidal disease is nowadays less frequently used, as it is not free from serious complications. Non-malignant tumours of the pharynx have been summed up in too few words. It seems futile to suggest the ligaturing of the superior laryngeal arteries for malignant disease of the larynx.

On the whole, the book is a credit to various writers, particularly to the authors of the sections on paranasal sinuses and the ear by A. Logan Turner and J. S. Fraser respectively, for these are probably the best descriptions in English to be found in so small a space. The practitioner will find that there is practically no subject in oto-rhino-laryngology about which some useful information will not be found in the book. We confidently recommend the volume to the general practitioner and to the student; the specialist will also find much pleasant and instructive reading.

ALEXANDER THE GREAT.

AN eminent radiologist of Turin, Mario Bertolotti, has undertaken the fascinating task of piecing together the fragmentary evidence of the medical life of Alexander III, known as "the Great". In his study, "Alessandro Magno",[1] he seeks to reconstruct the physical life of the warrior, to understand his character, to diagnose the nature and extent of his many wounds and to explain the manner of his death. It is not likely that every reader will agree with the author in all his conclusions, but the value of his work is undeniable and the manner of his writing beyond praise. With indefatigable zeal he has gathered all the available information and his verdict is given with judicial fairness. To be sure, the evidence is, at best, fragmentary, and after a lapse of twenty-three centuries speculation must be given free play.

The very birth of Alexander the Great is clouded with strange legends; for it is recorded how, on a night brilliant with stars, the magician Nettanebo, scrutinizing anxiously the heavens, counselled the parturient Olympias, that half wild and terrible visionary, to delay her travail lest a hybrid and incomplete monster should issue forth. The hours passed and the stars paled before the astrologist at last announced the conjunction to be favourable, and thereupon the groaning mother brought forth the fruit of her womb which, as it fell to the ground, produced an earthquake with loud thunder and terrible lightning: a prodigy had been born.

It is unfortunate that there exist no records from one of our own art who assisted at the royal bedside during his troubled and teeming life. He was early schooled in bitter warfare, and the story of his last eleven years is picturesque with heroic deeds from the Hellespont to Babylonia. He wore the aureole of many splendid victories, Granicus, Issus, Arbela, but, when at last he stood with the unknown world of the Ganges before him, the Macedonian army refused to proceed further.

Our notion of his personal appearance is gathered from contemporary writings, from monuments, medals and pieces of money. They reveal an athletic frame with a leonine head, held somewhat aslant in the posture made classical by the sculptor Lysippus. The cause of this wry-neck has ever excited the most interesting discussion. Was there a congenital torticollis or was the condition merely postural? The author quotes from Vasari who in his life of Michelangelo tells how greatly the artist suffered while painting the frescoes on the dome of the Sistine Chapel, which required him to stand for long periods with twisted neck and head. His vision was so much affected by the strained position that for months after Buonarotti could not read his letters or look at his drawings.

It is even more difficult to gauge the character of Alexander. It is accepted that he had a restless and impulsive energy and a vivid imagination, and that he could be terrible in anger. It is said he combined a passionate vehemence with sexual frigidity. After the murder of Clitus, struck down in a drunken brawl, as after the death of Hephæstion, we see him plunged in passionate mourning, the prey of a nervous crisis which only an attendant psychiatrist could have explained.

During his great adventure he suffered at least six serious wounds, of which a fractured base of the skull and a perforating wound of the thorax were the most severe. It was at the end of 329 B.C. that he was struck by a stone on the back of the neck and fell unconscious. There was no hæmorrhage from his nose or ears, but there was a temporary complete loss of sight. For long afterwards there persisted great weakness and a tendency to somnolence, which reveal the gravity of the cerebral commotion. His narcolepsy not infrequently alarmed his men, as when he remained in a long and profound sleep just before the fateful battle of Arbela. A dart piercing the left mammary region caused the perforating wound of his thorax. The lung itself was not pierced, but a secondary sepsis left behind a fistulous track.

But the hero of a hundred fights was not destined to meet death on the battlefield, leading the van or obstinately stemming a losing tide. At Babylonia in 323 B.C., under the hot sun of a torrid summer, he drew his last breath after an attack of what is believed to have been a malignant tertian fever. In these days we can only vaguely speculate on the immense part played by malaria in the fate of peoples, popes and kings.

Mute and weeping, the veterans of the army filed by the bedside of their dying leader, who looked at them through glazed eyes by the last gleams of his flickering consciousness. The son of Philip and Olympias, as Bertolotti writes, "*nel giro di pochi lustri bruciò la sua vita come una fiaccola*". Like a flare his life burned itself out briefly and luminously.

[1] "La Critica Medica Nella Storia: Alessandro Magno", by Mario Bertolotti: 1932. Torino: Fratelli Bocca. Royal 8vo., pp. 413, with illustrations.

A HANDBOOK ON SURGERY.

"A SHORT PRACTICE OF SURGERY", in two volumes, by Hamilton Bailey, F.R.C.S., and R. J. McNeill Love, M.S., F.R.C.S., is a valuable addition to the list of useful surgical publications.[1]

It comes from the pens of two experts in the art who have also the pens of ready writers. The text is fluent, the words are well chosen and the subject matter has been well selected. We have, therefore, a very readable survey of general surgery, suitable for the student preparing for examination and for the general practitioner who wishes to refresh himself on points in surgery.

Everything that is essential for the examinations for the lower degrees in surgery is included in these books, and everything the authors teach is thoroughly modern.

The authors, like the good teachers they are, follow the practice of giving the important matter in the ordinary type, the comparatively advanced, the less important matter and the rarer conditions in smaller type. They also follow the accepted practice in dividing the subject matter into anatomical regions and reference is, therefore, made extremely easy.

Illustrations are profuse, well selected and informative, the authors rightly claiming that a good illustration is better than much involved description.

We must congratulate the authors on their success in a very difficult task. They have given what is necessary to the part of the profession that they wished to serve, they have carefully preserved the balance of the book and have avoided all tendency to overloading.

The paper is good and the type clear and reading is freed from fatigue.

[1] "A Short Practice of Surgery," by H. Bailey, F.R.C.S. and A. J. McNeill Love, M.S., F.R.C.S.; Volume II: 1932. London: H. K. Lewis and Company, Limited. Demy 8vo., pp. 483, with 352 illustrations. Price: 20s. net.

The Medical Journal of Australia

SATURDAY, MAY 20, 1933.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

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Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

PATHOLOGICAL EXAMINATION OF TUMOURS.

TOWARDS the end of February of this year it was stated in one of the leading Sydney newspapers that about eighty doctors and scientists would meet at Canberra to hold the fourth Australian Cancer Conference. The journalist who penned the statement doubtless did not intend to be facetious; he probably meant to make no invidious distinction, but his general, though unintended, implication was true as far as some medical practitioners are concerned. This became apparent at the Cancer Conference when the classification of types of malignant disease was under discussion. This subject had been brought up for discussion at the previous conference in connexion with the medical certification of deaths from cancer. It is a difficult subject. On the one hand, for statistical purposes and for the advancement of knowledge, a detailed classification of tumours should be made, and all deaths from cancer should be grouped according to this classification. The attainment of this counsel of perfection involves detailed microscopic study of every tumour. On the other hand, medical practitioners with the best possible intentions are not always able to supply information which will allow a careful classification to be made. We do not propose to discuss the pathological classification of cancer at this juncture, but would state that medical practitioners, in certifying to the cause of death, do not always make full use of the information at their disposal. Further, they do not always satisfy themselves as to the nature of a tumour. This is either because they do not think that classification is important or because they are too lazy to bother about it. Whatever be the reason of their failure, the end result is the same. In any case they vindicate the Sydney journalist—medical practitioners are not all scientifically minded.

In this issue there appears an important paper by Dr. L. B. Bull and Dr. B. S. Hanson, of Adelaide. This paper is commended to the study of medical practitioners, particularly those in general practice. Attention should be directed to the discussion of these authors on biopsy and the pathological examination of tumours. Opinions would probably be divided as to how far routine biopsy in cancer was justified. Dr. Bull and Dr. Hanson have had a large experience at the Adelaide Hospital and they are quite definite in their pronouncement that no evil effects are likely to follow the removal of a piece of a tumour for examination. Their opinion must carry weight. Most medical practitioners will agree that manipulation of a tumour is more likely to cause dissemination of cancer cells than removal of a portion of the tumour with a sharp scalpel. When a section of a growth has been examined under the microscope by a competent morbid anatomist, doubt is in most cases replaced by certainty. A medical practitioner who is uncertain will subject the tumour to repeated manipulations, he will test its hardness time and time again and will move it about and keep moving it about to determine its attachments. He will do this because he is not sure. He wants to be sure. He ought to be sure. He cannot be sure unless he has the malignant process demonstrated to him under the microscope.

When a portion of a tumour or a whole tumour is sent to a pathologist, it should be accompanied by a full clinical history. Dr. Bull and Dr. Hanson are insistent on this point. Under ideal conditions the

pathologist, as well as the clinician, should examine the patient before any operation is undertaken, whether the operation be for removal of a piece of tissue or for removal of the whole tumour. When the pathologist has made his diagnosis the clinician should study both specimen and sections with the pathologist. In no better way can he extend his knowledge of pathology; and a clinician ignorant of pathology is like a mariner sailing an uncharted sea. In passing it must be stated that there are occasions on which the help of a radiologist rather than of a pathologist should be sought. For example, in a bone tumour in a child a diagnostic dose of X radiation may settle the question for all practical purposes. The pathological examination of tumours is a subject that should be discussed with that of the routine performance of *post mortem* examinations. Clinical diagnosis should be controlled by *post mortem* findings in every case of malignant disease. Some day an enlightened community may insist on *post mortem* examination after every death. Whether that day comes or not, the pathologist's collaboration must be regarded as indispensable to the diagnosis, and consequently to the treatment, of every neoplastic condition; in other words, he should take an equal share with the clinician in the investigation of every neoplasm.

Current Comment.

TRYPARSAMIDE AND ARSPHENAMINE COMPARED.

TRYPARSAMIDE is the sodium salt of the tryparsamide base, N-phenyl-glycineamide-para-arsonic acid, containing 24·57% of arsenic in pentavalent form. It was synthetized by W. A. Jacobs and M. Heidelberger in 1919, during the experiments of W. H. Brown and L. Pearce, who began with atoxyl and investigated the effects of various synthetized drugs in experimental trypanosomiasis. Tryparsamide was found to have a high therapeutic index and to be little toxic to animals. In human trypanosomiasis, including African sleeping sickness, its efficacy is undoubted and apparently the causative organisms are destroyed. It has been claimed that tryparsamide is curative in 100% of early cases in which the cerebro-spinal fluid is unaltered, but advanced cases are not all beneficially affected, and when the nervous system is involved the percentage of cures falls to 37·8%. Tryparsamide base may be given in tablet form, but in a tablet, twice the dose used for injection is necessary. By this method trypanosomes slowly disappear from the blood and cerebro-spinal fluid. The curative process is slow, but optic neuritis is not thus caused and the only toxic manifestations are slight diarrhœa and sickness. Tryparsamide is also stated to have given good results in epidemic encephalitis. When used in filaria the chyluria and lymphangitis are lessened, but no effect is exerted on the circulating microfilariæ.

H. C. Solomon, S. H. Epstein and A. Berk have written concerning the differential effects of arsphenamine and tryparasamide.[1] They state that arsphenamine has a very high therapeutic index as measured by its effects on *Spirochæta pallida*. Tryparsamide has very little effect on the organism in early human syphilis, but it exerts a very marked therapeutic effect on syphilis of the central nervous system, while arsphenamine has a very low index of efficacy. Arsphenamine has a very potent spirochæticidal effect both *in vivo* and *in vitro*. Its mode of attack is not clear. Ehrlich's "side-chain" theory does not afford an adequate explanation. Arsphenamine certainly kills the spirochætes in most tissues of the human organism, but it is not very effective when the organisms are well established in the central nervous system, as in paralytic dementia. In such cases its efficacy is very slight and it has been suggested that some hæmatocephalic barrier prevents the large arsenic molecule from entering the nervous tissues. But tryparsamide, which also contains the arsenic molecule, is much more effective in such cases. Probably there is no significant difference in the amounts of arsenic getting into the cerebro-spinal fluid when it is introduced into the blood stream in either form. Tryparsamide has little or no effect on early syphilis or syphilis with cutaneous manifestations. In 1923 W. F. Lorenz and his colleagues, having in mind the resemblance, clinical and pathological, of trypanosomiasis to syphilis of the nervous system, and considering that a drug efficacious in one might be expected to benefit the other, tried tryparsamide in syphilis of the nervous system and observed a definitely beneficial effect. Cases are described exhibiting the superiority of arsphenamine in syphilitic lesions of the skin and mucous membrane, and in gumma of bone, and its relative impotency to affect the nervous system; there are also reports of tryparsamide displaying much greater effectiveness on the nervous system but failing to prevent skin and mucous membrane relapses.

Additional evidence of the disparity in the effects of these two drugs is shown in sodoku or rat-bite fever due to *Spirochæta morsus-muris*. This disease responds well to arsphenamine and there is a rapid involution of the primary sore, which heals quickly. The skin lesions disappear and pyrexia abates. Under tryparsamide the primary sore heals rapidly, but the skin lesions and temperature are uninfluenced or the pyrexia may be aggravated. Arsphenamine attacks the organism itself, but tryparsamide has no effect upon it. It merely stimulates the

[1] *The American Journal of Syphilis*, January, 1933.

activity of local tissues about the open primary lesion and expedites their healing without destruction of the spirochætes in the blood stream. How this stimulation is produced is not clear. The difference between these two drugs is clearly indicated also in malaria. Arsphenamine is definitely plasmodicidal. In inoculation malaria it promptly clears the blood of plasmodia and terminates the fever. Tryparsamide has no effect on the plasmodia or the pyrexia. Arsphenamine is the more efficacious in early and late systemic syphilis with cutaneous, osseous and mucous membrane lesions. It is both spirochæticide and plasmodicide. Tryparsamide has no lethal effect on spirochætes or plasmodia and in syphilis is most efficacious when the central nervous system is implicated. The explanation of its beneficial effect is not clear. In some way it stimulates local tissue and possibly, as in local benefit in sodoku, so in syphilis of the central nervous system tryparsamide has a local action within the local areas of the nervous system.

Unfortunately this explanation is not very illuminating and leads nowhere. Equally nebulous are the suggestions that this drug acts as a tonic and stimulates resistance and the defensive mechanism. It certainly has a remarkable power of penetrating the central nervous system, far more so than arsphenamine. It readily gains access to the cerebro-spinal fluid. It is said to induce resolution and healing of syphilitic processes even in the presence of active spirochætes, which it does not destroy. It has been shown in rabbits that after tryparsamide administration the parenchyma of the brain and spinal cord contains definite amounts of arsenic. It is excreted rapidly by the kidneys and by the whole intestinal tract. After the administration of a lethal dose the liver takes up an increasing amount of arsenic, the extent of liver damage apparently being parallel to the quantity taken up. There are definite dangers attending its therapeutic administration; these are not mentioned by Solomon and his colleagues. Like other pentavalent arsenic preparations, it may induce toxic jaundice and amblyopia. It is very apt to induce optic neuritis and atrophy. There may be recovery from visual disturbances, but in a minority of cases these are permanent. Careful ophthalmoscopic examination is necessary before and during treatment, and any history of disordered vision is an indication for immediate discontinuance. The danger signals are dimness of vision, fickering sensations and flashes of light with contraction of the visual fields and atrophic changes in the disks. Hazards to the optic nerves have been estimated at 4·3%. Much diversity of opinion exists as to which syphilitic manifestation is most benefited by tryparsamide. Paralytic dementia and meningovascular syphilis, however, are said to respond best. As regards tabes, opinions are most conflicting. The optimistic reports from America are not confirmed in England. It is generally agreed that the response to the Wassermann test is commonly uninfluenced. Many observers advise the administration of tryparsamide in conjunction with other forms of treatment, such as fever therapy and bismuth injections; or its efficacy may be enhanced by lumbar puncture and spinal drainage. W. S. Dawson formed the opinion that in no case was any remarkable result effected by tryparsamide which might not have followed the use of other arsenical compounds. Tryparsamide is obviously no panacea, but may be of some use in syphilis of the central nervous system, if its danger to the optic nerve be carefully regarded.

FILARIASIS

THERE are many details in the life history of *Wuchereria bancrofti* (formerly called *Filaria bancrofti*) that are not yet known. Practically nothing is understood of the causes of periodicity and non-periodicity in the appearance of microfilariæ in the peripheral blood; very little is known of the duration of the worm's life in the human body; and the manner in which filarial infestation produces pathological lesions is as yet only conjectured. Knowledge is gradually being increased, particularly by workers in India, of whom H. W. Acton and S. Sundar Rao deserve special mention. Sundar Rao has recently published some notes concerning the possible duration of life of *microfilaria bancrofti* in the human body.[1] He gives brief histories of two patients who received treatment for filariasis in the Carmichael Hospital for Tropical Diseases. The first patient had a cyst on the left elbow; it contained fluid in which innumerable microfilariæ were seen. The peripheral blood also contained microfilariæ. The cyst was removed by operation and was found to contain five adult worms. Microfilariæ could be found in the peripheral blood until between two and three months had elapsed after the operation, and could never be found thereafter. The second patient also suffered from cysts containing fluid and microfilariæ. These cysts also, when removed, were found to contain adult worms. Microfilariæ were not found in the peripheral blood until after the operation; they were then observed at each daily examination for some time; later they became fewer in number and could only be found occasionally. Daily examination was continued for 107 days; but no embryo was found after the seventieth day. Sundar Rao remarks that in each case the worms contained in the cysts were apparently the only source of microfilariæ. As microfilariæ could not be found in the peripheral blood after the lapse of 70 days, it may be assumed that they died out and that 70 days represented the life span of the youngest of them.

Sundar Rao's observations are of interest, and may prove to be important. They do not give conclusive evidence of the duration of life of *microfilaria bancrofti* in man's body; but they provide a useful indication of one line along which the study of the life history of *Wuchereria bancrofti* may be pursued. Australia has been associated with one great advance in the knowledge of filariasis; there is great scope here for further research.

[1] *The Indian Medical Gazette*, January, 1933.

Abstracts from Current Medical Literature.

PÆDIATRICS.

Lipoid Histiocytosis.

C. F. FISHER (*Archives of Pediatrics*, September, 1932) outlines the chief features of lipoid histiocytosis or Niemann-Pick's disease, a liver-spleen disease first described by Niemann in 1914 and separated by him from Gaucher's disease. Pick showed that it had an early onset, that it occurred in Jewish children, and was characterized by enlargement of the liver and spleen, retarded physical and mental development and a flooding of the reticulo-endothelial system with foam cell forms of histiocytes. Death usually occurs before two years, from cachexia in infection. Other clinical signs described include motor weakness, general adenopathy, mild secondary anæmia, hyperlipoidæmia, and a brownish-yellow discoloration of the skin. Mental changes and the presence of a cherry red spot in the macula render differentiation from amaurotic family idiocy difficult. They are said to occur often together. Irregular fever may also occur. The urine is normal, and in the blood there occurs a moderate anæmia with leucocytosis and the characteristic vacuolation of the non-granular cells. There is no change in the bleeding or coagulation time. Great increase of the phosphated fraction of the blood lipoids distinguishes the disease from Gaucher's disease, and diagnosis is usually based on the finding of typical foam cells on splenic puncture. According to the author, only twenty-five undoubted cases have been reported. He describes another, giving no abnormal family history, which complied with the above criteria. The skeleton was radiographically healthy and the fundi had the macular coloration. The blood count had certain points of resemblance to lymphatic leuchæmia. The child failed to respond to treatment consisting mainly in the administration of a high vitamin diet.

Patent Ductus Arteriosus.

D. C. MUIR AND J. W. BROWN (*Archives of Disease in Childhood*, December, 1932), while admitting patent *ductus arteriosus* to be the most completely studied lesion in congenital cardiac pathology, have investigated a further twenty cases occurring in a series of eighty-eight instances of congenital heart disease. Symptoms are rare. The subjects are usually pale and slightly built. Cyanosis is usually absent. Fits may occur, especially in infancy, when the act of sucking raises the pressure in the pulmonary artery above that in the aorta and causes venous shunting. The most characteristic physical sign is a long, rough murmur heard close to the sternum in the first or second left interspace. The character of the murmur varies with the age of the patient and only becomes continuous with the full development of the pulmonary artery in later childhood. The murmur increases on inspiration and is usually best heard with the patient erect. A diastolic bruit may be heard along the left edge of the sternum, and there is undue accentuation of the pulmonary second sound, to be sought below the area of maximum intensity of the murmur. A systolic or diastolic thrill is present in about 25% of the cases. Gerhardt's dulness is much more frequently found in adults than children. It is due to dilatation of the pulmonary artery. The diastolic pressure may be lowered, causing confusion with aortic incompetence. The pulses may be unequal; in this case the left is smaller than the right. *Pulsus paradoxus* and paralysis of the left vocal cord have been described infrequently. Radiographic screening shows dilatation of the pulmonary artery, best seen in the right oblique position; but the appearances are normal, despite well marked clinical signs. There is nothing in the electrocardiogram in any way diagnostic of the condition.

Gold Tribromide in Pertussis.

J. EPSTEIN (*Archives of Pediatrics*, January, 1933) states that the result of treatment of seventy-five patients with pertussis by means of gold tribromide was most gratifying. The effects even in the established cases were noticed after two or three days, when the cough was less frequent, the attacks shorter and milder, and the sleep less disturbed. The drug is a neutral salt of gold and hydrobromic acid, is brownish-black in colour, and contains about 45% of gold. The clinical effect is supposed to result from a compound which has both a neurosedative and an antibacterial action. It is given in aqueous solution by mouth, a teaspoonful three or four times a day and at night. Each dose contains from 0·0032 gramme to 0·0065 gramme (one-twentieth to one-tenth of of a grain). The drug can also be used with profit as an inhalant, one drachm of an elixir of gold tribromide to an ounce of water being used.

Treatment of Pyknolepsy.

JOHN ZAHORSKY (*Archives of Pediatrics*, January, 1933) discusses the diagnosis of pyknolepsy and the difficulty in distinguishing it from *petit mal*, except by the fact that the symptoms are wholly uninfluenced by bromide medication, and that there is no change in mentality or character. The disease is generally regarded as being distinct from but related to narcolepsy, and patients have been reported as exhibiting certain symptoms common to each disorder. A neuropathic constitution was found in all cases, but most careful analysis of possible causes and of the symptomatology has failed to reveal any constant feature which is common to the past history or physical constitution of these patients or which may serve to differentiate the condition from minor epilepsy. Search has been made for a therapeutic test which may decide the question of diagnosis, since the bromide test, already referred to, is not wholly reliable. Patients have been reported to have shown improvement on an anti-ketogenic and a ketogenic diet. Meyer reports a reduction in the number of attacks following the use of calcium lactate and small doses of phenobarbital. Atropine has been said to succeed in some instances. Seizing on the possible relationship to narcolepsy, Zahorsky has been giving ephedrine sulphate to a series of sufferers from pyknolepsy. He uses a dosage of 0·024 gramme (three-eighths of a grain) twice daily. Given to patients with evident minor epilepsy, he says it seemed to aggravate their condition.

Pulmonary Changes in Rheumatic Pericarditis.

G. T. COOK (*The British Journal of Children's Diseases*, October-December, 1932) has studied thirteen cases of rheumatic pericarditis in childhood and found pulmonary signs in twelve. In seven of the twelve cases the signs were confined to the left base. In more than half there was not more than three days' difference between the onset of the two conditions. There seemed to be an association between the persistence of the pericardial signs and those arising in the lung. A pericardial effusion was diagnosed in five of the thirteen cases, and a pleural effusion in two. Concerning *post mortem* evidence, Cook has reviewed five cases in which death was due to rheumatic pericarditis. Fibrinous pleurisy was present in four of these cases, with free fluid in the pleural sac. Collapse of the lower lobe was present in every instance, and an early stage of bronchopneumonia in two. Microscopic examination of the lungs of these patients showed a considerable degree of inflammation in the alveoli, suggesting early rheumatic pneumonia. The author finally notes that the pulmonary signs occur much more frequently on the left side than on the right, and that the pulmonary condition can occur quite apart from a pleural or pericardial effusion. He suggests that the collapse observed may be a reflex active massive collapse occurring as a vagal reflex following pericarditis.

Osseous Dystrophy Following Icterus Gravis Neonatorum.

FRANCES BRAID (*Archives of Disease in Childhood*, December, 1932) records a case of great rarity in which two very unusual conditions, *icterus gravis* and osseous dystrophy, have occurred in the same child. The second case took the form of a cystic condition of all the long bones, which, in consequence, underwent some curvature and deformity. Blood calcium and phosphorus contents were within normal limits, as was also fat

excretion. The blood urea content was within normal limits, and the blood did not react to the Wassermann test. Treatment usually given for rickets, administration of glandular or liver preparations, have been of no avail. The author states that the dramatic improvement in patients suffering from familial *icterus gravis*, when treated with maternal serum or whole blood, is often not maintained during the subsequent development of these infants. A profound disturbance of the central nervous and muscular systems may follow, and, apparently, of the osseous system also. The author thinks that the calcium disturbance originates in the liver and that derangement of obscure functions of this organ controlling vitamin stores forms the link between the two disturbances manifested in his patient. He attributes the lack of other cases in the literature to the fact that survival from *icterus gravis* has been rare until recent years.

ORTHOPÆDIC SURGERY.

Pathological Fractures.

E. L. ELIASON (*Surgery, Gynecology and Obstetrics*, February, 1933) classifies causes of pathological fracture as: (i) local lesions, which comprise benign and malignant tumours, acute and chronic infections, and chemical and pressure causes; (ii) fragility due to general disease; (iii) fragility due to hereditary disease. He discusses union and non-union in these fractures, and considers that non-union of various grades to excessive union may occur in different types of pathological fracture or in the same disease. The vast majority of pathological fractures unite; union may occur in 89% of the different diseases, and 20% may have excessive callus production. Delayed union (but union nevertheless) may take place in 70%. In only 16% is non-union the rule, though Bloodgood is aware of its occurrence in sarcoma, hypernephroma and endothelioma. Ten *per centum* of this latter group are placed in the "little known class", as insufficient data on reported cases made this necessary. He summarizes his findings as follows. In fractures due to benign tumours, union is the rule; in cysts, the fracture episode usually results in a cure of the cystic condition; in malignant tumours, union often occurs. Hawley states that in carcinoma union is the rule. Bloodgood states that in metastatic carcinoma union rarely takes place. In Pancoast's experience 40% of pathological fractures due to carcinoma unite with or without irradiation. In sarcoma Bloodgood states that union is almost unheard of. Union is known to have occurred in malignant myeloma. Primary bone tumours are complicated by fracture in 22·7% of cases. In acute and subacute inflammatory conditions union is the general end result, if the infection has early and adequate surgical treatment. In neglected cases, especially in adults, non-union may occur. In chronic inflammatory conditions union is the rule, with excessive callus formation. In fractures occurring in general disease, union is delayed or absent, depending upon the course of the general disease. In rickets, osteomalacia and scurvy, proper treatment results in union; osteomalacic fracture heals with excessive provisional callus.

Intracapsular Fractures of the Neck of the Femur.

LAURENCE JONES (*Annals of Surgery*, February, 1933) describes a method of inserting a double-ended screw into fractures of the neck of the humerus. After placing the thigh in abduction he drills two Kirschner wires through the trochanter to fix the head and neck while he inserts a screw with a grooved head and a burred end into the neck through the trochanter. The outer end of this screw is provided with a thread to receive a flanged screw; by screwing the latter on the former the fractured surfaces are compressed together. After four months the burr of the proximal end of the screw was still tightly engaged in the head and was removed under local anæsthesia. He considers that convalescence is hastened and that ultimate function is better with this method than with the other recognized methods.

Ganglionectomy.

W. MCK. CRAIG AND J. W. KERNOHAN (*Surgery, Gynecology and Obstetrics*, April, 1933) report that fifty persons suffering from Raynaud's disease were submitted to operation. On some the operation of bilateral lumbar ganglionectomy, on others bilateral cervico-thoracic ganglionectomy, and on others, both operations, were done. In almost all the cases in which operation was performed, the relief of pain was almost instantaneous, the cyanosis disappeared, and the skin became warm, pink and dry; the ulcers healed, the nails took on normal growth, and the patient was restored to normal health. The walls of the blood vessels of the ganglia, examined histologically, were seen to be thickened; the lumen was narrowed, so that the ratio of lumen to wall, which is normally 2 to 1, in small arteries was now only 1·7 to 1. In addition, in a few arterioles there was an increase in prominence of the lining endothelial cells; but this was by no means universal, and was only mild. There was no evidence of acute or chronic inflammation; there were no fibroblasts or increase in the connective tissue as shown by the Van Gieson stain, and polymorphonuclear leucocytes were not present. Although there was no inflammation, lymphocyte-like cells were present; these were in two small groups, and not scattered diffusely throughout the ganglia. There were 97 patients with *thromboangiitis obliterans* on whom the cervico-thoracic or lumbar ganglionectomy or both types of operation were performed. The ganglia removed were studied histologically. The authors consider that by means of operations on the sympathetic system it is possible to relieve the vasomotor spasm of the collateral vessels, which improves circulation, tends to prevent ulceration, infection and gangrene, and hastens healing of existing ulcers and abrasions. In suitable cases, selected by means of the various tests of altered vaso-constriction, the temperature of the skin is increased from 1·1° to 5·5° C. (2° to 10° F.), depending on the amount of vasoconstriction present before the operation; the ulcers begin to heal, and the patient is restored to his former status as a wage earner. Histological study of the ganglia showed that there was more proliferation of the lining endothelial cells of the arterioles and small arteries than in Raynaud's disease, but this proliferation was by no means universal. There was also slight thickening of the walls of the larger vessels of the ganglia, so that the normal ratio of lumen to wall (2 to 1) was reduced to 1·8 to 1, a reduction not so pronounced as that seen in the vessels examined in cases of Raynaud's disease. Ganglia from 46 patients with arthritis were studied histologically. In some cases both upper or both lower extremities were denervated, and in others all four extremities. In certain selected cases of the arthritic group operations on the sympathetic ganglia are followed by relief of symptoms and by the improvement in circulation which follows the vasodilatation. The skin becomes warm and dry, and pain is generally relieved; the tenderness tends to disappear, and the swelling subsides. The changes observed histologically in these ganglia were similar to those in the ganglia removed from patients suffering from Raynaud's disease and *thromboangiitis obliterans*. Most of the blood vessels were normal, but in a few there was mild proliferation of the lining endothelium of the intima, and in others there was slight thickening of the walls of the larger arteries in the ganglia. The ratio of lumen to wall in this group was 1·8 to 1, instead of the normal 2 to 1. There was no evidence of acute or chronic inflammation; polymorphonuclear leucocytes were not present, fibroblasts were absent, and there was no increase in adult connective tissue. Although there was no inflammation, there were small collections of lymphocyte-like cells; but these were collected in groups and not scattered diffusely throughout the ganglia. The authors consider that their various observations lend support to the conclusion, which has frequently been advanced, that the noxious agent causing the vascular diseases for which operative relief has been instituted, does not act on the sympathetic ganglia. Since the removal of the sympathetic ganglia leads to improvement of the blood supply to the affected limbs, it would seem that the ganglia act simply as relay stations for impulses from higher centres, where the disease originates.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE SOUTH AUSTRALIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Anatomy Lecture Theatre, University of Adelaide, on February 23, 1933, DR. ST. J. POOLE, the President, in the chair.

Varicose Veins and Ulcers.

DR. A. DAWKINS read a paper entitled: "Varicose Veins and Ulcers: Their Ætiology and Treatment" (see page 603).

DR. A. BRITTEN JONES congratulated the speaker on his excellent paper. With regard to the direction of blood flow in varicose veins, he considered that it was reversed in the larger varieties of varicosities. In support of this opinion, he mentioned that he had noticed, when injecting some veins with the patient in the standing position, a contraction of the vein wall occurred for some distance below the injection site, but not above it. He had seen several cases that failed to respond to any of the sclerosing solutions, and in these he was in favour of the method of tying the internal saphenous vein just below the saphenous opening, and later injecting the veins. He had not seen a case that had failed to respond to this procedure.

DR. P. S. MESSENT drew attention to the experiments carried out in America whereby radio-opaque material was injected into the varicose veins and its progress was watched on the screen. The direction of the flow was found to depend on gravity. In regard to varicose ulcers, he considered that Unna's paste, if properly applied, was quite as satisfactory as elastoplast. He also stated that he did not believe that all incurable ulcers were necessarily syphilitic.

DR. IAN HAMILTON discussed the dangers of infection and secondary hæmorrhage in injection ulcers. He advocated a method of partial sclerosis of big veins, obtained by applying pressure along the length of vein after the injection. He also advocated perivenous injection for some cases.

Dr. Dawkins, in reply, stated that he had never purposely injected the perivenous area, but one or two accidental cases had responded to this method. But, owing to the ease with which ulcer formation followed accidental perivenous injection, he hesitated to recommend the method.

DR. D. R. WALLMAN also advocated the application of pressure to the injected segment of vein by means of a pad and bandage.

DR. G. M. HONE quoted a case in which ulcers formed at the sites of injection several weeks after the treatment had been given.

In reply, Dr. Dawkins said he had not seen any similar cases and was unable to comment.

DR. F. H. MAKIN said he was glad to see surgeons entering a field which had long been considered that of the dermatologist, and he welcomed their assistance. He remarked that he had found elastoplast more satisfactory than Unna's paste.

DR. BRIAN SWIFT agreed as to the inadvisability of carrying out injection treatment during pregnancy. He referred to garters as a possible predisposing cause.

Dr. Dawkins replied that, although he considered garters might aggravate varicose veins already present, he thought it unlikely that they could play any real part in the original formation of the varices.

DR. OWEN MOULDEN reported a case of spontaneous sclerosis of a *saphena varix*, and stated that he had found Unna's paste more efficient and satisfactory than elastoplast.

Tetanus in a Child.

DR. C. DUGUID gave notes of a case of tetanus in a child of two years and three months.

On December 29, 1932, a twig of a peach tree caught in the canal of the right ear of a child while playing in the garden. On January 1 the twig was removed under an anæsthetic. At 8.30 p.m. on January 11, when called to attend her sisters, Dr. Duguid noticed a suspicious grin on the face of the baby, and a trained nurse was put in charge to note developments. At 3 a.m. on January 13 the child had its first severe spasm. Tetanus was diagnosed, and Dr. Le Messurier was called in consultation, and Dr. W. Sangster to treat the ear. At 9 a.m., under an anæsthetic, the ear was opened and 30,000 units of tetanus antiserum were injected intramuscularly. At 9 p.m. a further 20,000 units were given. The child was given 0·6 gramme (10 grains) of chloral every four hours by mouth for ten days, the amount being then gradually diminished. On January 14 only 20,000 units of serum were given, but, because of the increased severity of the attacks, 20,000 units were given night and morning for the next three days, then 20,000 daily for six days—in all, 310,000 international units. All antitoxin was given intramuscularly.

In addition to the darkening and quietening of the room, the patient had to have the shoulders propped up for ease of breathing.

The worst attack was on January 14, after which the child was cyanosed and unconscious for two minutes, and very ill for the next twelve hours, with almost uncountable pulse, and very rapid and shallow respiration. Brandy was administered at this period.

Feeding was chiefly by milk and egg flips. It was always possible to get these between the teeth.

Dr. Duguid said that he had to thank Dr. Dwyer, of the Adelaide Hospital for sharing his experience of these cases with him, and for his confirmation of his preference for the intramuscular route. One school advocated intrathecal and intravenous injection at the beginning; the other school advised against both. There had been a number of cases of "aseptic meningitis" from the former method, and fatal anaphylaxis had followed the latter.

The patient went home on the sixteenth day. Thereafter only occasional doses of chloral were given at bedtime, because of extra restlessness. In a month's time the patient was practically normal.

A MEETING OF THE NEW SOUTH WALES BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at Broughton Hall, Sydney, on April 20, 1933. The meeting took the form of a series of clinical demonstrations.

Manic-Depressive Psychosis.

A man, fifty years of age, was shown. There was a long history of excessive indulgence in alcohol. For some time ill-feeling had existed between him and fellow workmen because they had accused a foreman of malpractice and he had been suspended; the patient was very friendly with this man and he resented their action. He had begun to worry about it, and had become very depressed and could not sleep. In addition he had found that he was incapable of doing any strenuous work, because of a urethral fistula, the result of an old injury. He was afraid to climb to any height for fear of collapsing. For two years he had become increasingly seclusive, shunning company and brooding over his troubles. Prior to this he had been boisterous and cheerful. A few days before admission to hospital his mood had changed and he had become cheerful, talkative and mildly elated. At the time of admission the manic state was well pronounced. The patient was restless and noisy and continually calling out in a loud tone. He gave a rambling account of his illness and was garrulous, argumentative and abusive. At times he became rather emotional, and, although stating that he was very apprehensive, he was aggressive and threatening in manner towards other patients. His physical condition was good except for the presence of pus in the urine and a urethral fistula. There was some slight exaggeration of the deep reflexes.

His previous health had always been good, and there was no history of any family tendencies. He did various odd jobs until nineteen years before admission, when he obtained permanent work. He had always been a heavy drinker. Temperamentally he was hasty, irritable and impulsive, and normally was loud-voiced and argumentative.

His condition remained hypomanic in spite of the use of sedatives and hot baths of one hour's duration, twice a day. He was restless, elated, expansive, euphoric, noisy and exacting. In spite of his threatening and abusive manner, at times he was quite amenable and manageable with tact.

A second patient, suffering from manic-depressive psychosis, was an unmarried woman, aged twenty-five years, who had been admitted ten weeks previously in a state of acute mania.

Her mother had suffered from recurrent attacks of melancholia throughout her life, in one of which she had been certified insane and admitted to a mental hospital; a maternal uncle was morose, and other members of the mother's family were subject to attacks of depression.

The patient was reared by a paternal aunt from the age of six years. Her education was irregular; but she was above the average intellectually, and had a special flair for writing. She had suffered her first "breakdown" when she was twenty-one years old, and a second at twenty-three years; both attacks had been brief.

For some months the patient had not been feeling well; she had been depressed and suffered from insomnia and had difficulty in doing her work. When admitted to hospital she appeared a little elevated and talkative, but seemed to have a considerable measure of self-control. The same night, however, she became very restless, destructive, and noisy, and stripped her clothes and exposed herself. A condition of acute mania continued for a week, characterized by noisy talking, shouting, stripping her clothes and rolling and tumbling about her room. During this period she was treated beneficially with continuous warm baths. She eventually became quiet and orderly, and was placed in a dormitory with other patients. She still manifested a press of activity in the form of constant letter writing.

A week or two after transference to a ward she became mildly depressed, listless and dull. This phase, which lasted for a fortnight, culminated in several hysterical episodes of screaming, following which she again became manic, reproducing her former symptoms, and, in particular, running around her room, turning somersaults, and rolling off her bed. This state continued for two or three weeks, when she again regained control and remained quiet and disinterested, but apparently not depressed.

Traumatic Neurosis.

Hysterical Spasm of Erector Spinæ Muscles.

A man, aged thirty-nine years, suffering from hysterical spasm of the *erector spinæ* muscles, was shown. He was born in Jugoslavia, and had been in Australia about five years. In January, 1929, a beam of timber, forty feet long, fell from a building and struck him on the back, knocking him down. He was unconscious for a brief period and recovered to find that he had a severe pain in the mid-lumbar region. He was removed to hospital, and remained for six weeks between sand-bags and then put in plaster of Paris, extending from the axillæ to the hips, for one month. When this was taken off, he was measured and supplied with a celluloid corset, which he had worn ever since. In spite of this, the pain in the back persisted; it was aggravated by movement. He had received treatment by hot salt baths, massage, liniments and electrotherapy, without benefit. He had been paid compensation for his accident until May, 1930.

He was quite alert at the time of admission, and able to give a good account of his illness. He was intelligent, bright and pleasant, although extremely hypochondriacal, complaining of pain and stiffness in the lumbar region and holding his back rigidly. He made no effort to relax the dorsi-lumbar muscles, which were on guard the whole time. He bent forward *en bloc*. There was marked tenderness over the third and fourth lumbar vertebræ, and to a less extent over the sacrum and the left iliac crest. X ray examination revealed fractures of the transverse processes of the third and fourth vertebræ on the left side. There was no sign of union. His condition otherwise was quite satisfactory, the deep reflexes being normal and other systems clear.

He remained quite cheerful in spite of his discomfort, but continued to hold his back rigid, in spite of treatment by ionization with potassium iodide, massage, and exercise. He was encouraged to discard his supports and walking stick, but still walked with great care and made no effort to relax the *erector spinæ* muscles. He was discharged from hospital, but readmitted eighteen months later, when it was found that he had lost considerable weight, and had been sleeping badly. The pain in the back was much more severe, and he had returned to the use of the celluloid support. At the time of the meeting his general condition was improving once more; but he walked with difficulty and the spasm of the back muscles continued.

Hysteria.

The next patient suffering from traumatic neurosis was a boy, aged seventeen years, who had been struck on the neck with pipes while at work nine months previously, and had been unconscious for more than two hours. He had been taken to hospital, where he remained for three weeks. After this he became subject to "turns", in which he was giddy and had singing noises in his ears, and the ground seemed to come up towards him. He fell and lost consciousness during the "turns", recovering to find people holding him. He had never bitten his tongue or been incontinent during a seizure. The day following his return from hospital he had an attack, fell downstairs, and was taken back; but after a short stay he was advised to resume his work as a picture framer. He did this, but had to give it up after two days on account of his frequent fits. His compensation payments were discontinued; but this did not hasten his recovery. While at home convalescing, he went for picnics and would wander away, to be found later by other members of the party, lying on the ground, in a "turn".

He was admitted to the Coast Hospital in January and remained there for two months. X ray examination of the skull revealed no abnormality; but lumbar puncture revealed that the cerebro-spinal fluid was under increased pressure, and contained 79 milligrammes of sugar and 720 milligrammes of chlorides per one hundred cubic centimetres. There were no cells or organisms present, and the fluid did not react to the Wassermann test. His fits were observed closely, and it was seen that he did not pass urine, bite his tongue, or injure himself in any way, during them. He became somewhat violent and threw his arms about, but could be roused, and would talk in a surly, monotonous manner, when the seizure terminated, generally after about a minute. He remained drowsy for twenty-four to forty-eight hours after, and was then apparently normal until the next attack.

At the age of three years he had been struck on the head with a horseshoe and taken to Sydney Hospital. At eight years he had had concussion, and twelve months before admission he had been thrown while wrestling and had become unconscious. Except for these accidents, his previous health had always been good, and he was apparently a normal, bright, moderately intelligent boy. He had been working in a picture framer's for three years and was interested and contented there.

There was no history of any form of nervous or mental disorder in the family.

At the time of admission he complained that his neck was very sore and that he had severe pains behind the ears and was afraid that he might be developing acute mastoid disease. His general condition was good. The reflexes were normal. The only abnormality found was the presence of pus in the urine, which was alkaline. The blood pressure was rather low, the systolic pressure being 108 and the diastolic 80 millimetres of mercury.

He had no seizures after admission; but, for a short period, he was dull and indifferent, and in a condition somewhat similar to post-epileptic confusion; otherwise he was alert, cheerful, and perfectly amenable.

Hysterical Spasm of the Arm.

A third patient suffering from traumatic neurosis was a tram conductor, aged fifty years, who had fallen from a tram car while collecting fares eighteen months previously. The tram was moving at the time. He fell backwards, striking his head on the ground. He was unconscious for

twenty-two days and was not expected to recover. His condition was diagnosed as fracture of the skull and cervical vertebræ. On regaining consciousness, he had shooting pains in the head and a tendency to fall or roll on his right side. After ten weeks in hospital he was discharged, and, although weak, he remained fairly well for two months, and then developed jerky, convulsive movements of the body generally. This persisted for seven months. He was sent for by the Railway Department and had a "fit" in the office, losing consciousness. There were three such attacks in all. He gradually improved, but was unable to do any arduous work because of weakness. About nine months before admission his right arm began to jerk in an irregular fashion; this condition was still present when he was admitted to Broughton Hall. At times he became very emotional and would weep for hours for no obvious reason. If he was subjected to a sudden shock, both arms would jerk upwards, and, if the shock was particularly severe, he would clap his hands together involuntarily. He was frequently the cause of alarm to passengers on the tram cars because of his sudden movements and exclamatory noises, as he gave them the impression that he was about to strike out at them. His previous health had always been good and he had successfully carried out his duties for fourteen years.

When admitted to Broughton Hall he was quite bright and cheerful, in spite of his condition. He complained of weakness and difficulty in concentrating. At times his speech was remarkably explosive. There were persistent, purposeless movements, of a defensive nature, of the right arm, associated with an exclamatory grunt. He had a good measure of insight into his condition and expressed a desire to get well. His physical condition was normal.

He settled down satisfactorily in a short time and improved considerably with rest and encouragement. He exhibited rather childish delight in recounting his various symptoms to all and sundry, and expressed undue anxiety to assist in the treatment of other patients. The movements disappeared, and he was able to work quite diligently, although somewhat officiously, in the grounds. He occasionally became upset because of some trifling matter and reacted with a recurrence of his symptoms and an emotional outburst, or even a fit. His lack of confidence and his dependence on others were apparently the strongest factors militating against his immediate recovery.

Cerebral Syphilis.

A man, aged fifty-five years, suffering from cerebral syphilis, was shown. He had contracted syphilis twenty-eight years previously. He had received treatment for two years and had then been pronounced cured; but eleven years before admission he developed a hemiplegia of the right side, which had remained weak ever since, although he had managed to carry on his work as an orchardist. Six weeks before admission, while sitting down one night, he felt giddy, and had a tendency to fall backwards. The left side became powerless, his speech became slurred, and he felt a tingling in the left side of his face and tongue. He remained in bed for two weeks at home and was then admitted to the Royal Prince Alfred Hospital. He recovered some power in the left arm, but the leg remained affected and he dragged it when walking.

His mental condition was quite good at the time of his admission, and he was alert and able to give a full, coherent and intelligent account of his illness. There remained a slight impairment of speech; otherwise he appeared normal. Vision was impaired, but had been so since childhood. There was a coarse nystagmus when the patient looked to the left. He had suffered from progressive deafness in both ears for fourteen years. There was left facial paresis. Sensation was normal on both sides; but there was left-sided paresis, with some increased tonicity and a tendency for the affected leg to be dragged. Rombergism was present. The knee jerks were more pronounced on the right side; the plantar responses were flexor on this side. The pupils were small, irregular and sluggish in reaction to light. No abnormality was found in other systems. The blood did not react to the Wassermann test. The cerebro-spinal fluid reacted slightly to the Wassermann and Boas tests.

The patient remained quiet and well conducted after admission. He was inoculated with malaria by the intramuscular injection of one cubic centimetre of blood from a malarious patient.

The second patient suffering from cerebral syphilis was a man, aged forty-nine years. He admitted having sustained luetic infection in 1916, while in hospital in England suffering from hemiplegia, the result of a mine explosion in Gallipoli. On return to Australia he was sent to Milson Island; but he denied that he received any anti-syphilitic treatment there. He was admitted to the Military Cottages at Callan Park; but his mental state was such that he had to be certified as insane. At this time he was depressed, confused, dull and stupid, and his speech and memory were affected. He was discharged in 1917, and worked at Garden Island until 1929, when he began to have seizures at intervals of three or four months, and he was discharged. The fits were epileptiform in nature, with clonic and tonic stages and loss of consciousness. They became more frequent, and in the intervals between them he had periods of confusion and incoordination. He had become very dull and disinterested during the few months prior to the meeting.

He was quiet and amenable on admission, with impairment of both recent and remote memory. Although he retained some insight into his condition, he was dull, indifferent, and rather apathetic and anergic; there were some slurring of speech and considerable retardation of ideation. His pupils were unequal, the left being larger than the right, and they reacted sluggishly to light. There were left-sided facial paresis and a coarse tremor of the tongue. Sensation was impaired, and paresis and rigidity were present on the left side of the body. Rombergism was present. The knee jerks were exaggerated. The blood and cerebro-spinal fluid did not react to the Wassermann test.

After admission he became rather foolish and childish in demeanour, but perfectly amenable. He had two typical seizures and remained mildly confused for some hours afterwards. No alteration occurred in his mental state. It was remarked that a long-standing dementia of this type was not likely to improve under any treatment at such a late date.

Dementia Paralytica.

The next patient was a man, aged forty-seven years, who had been treated by malarial infection for *dementia paralytica*. He had been infected with syphilis twenty-five years previously. He was a bank manager, and had carried on his work successfully until seven years previously, when he rather impulsively resigned his position, because of a "nervous breakdown", in which he was depressed and emotional. He apparently recovered after about four months, but was unable to obtain constant work; all that he had done was debt collecting for a few months. During the eight months before admission he did no work, but seemed quite well until two weeks before admission, when he became very active, gardening energetically, making and remaking his bed, and behaving in a generally foolish and irresponsible fashion. He became very obstructive and obstinate, and refused to do anything he was asked to do. When being brought to hospital he made a great display of weakness and had to be assisted.

At the time of his admission to hospital his symptoms were more suggestive of an acute anxiety state than an organic dementia. He was restless, emotional, tearful, and extremely agitated and apprehensive. He grimaced, moaned, threw himself on the floor, and at times was aggressive and threatening in manner. He did not cooperate at all. It was almost impossible to examine him. The following day he settled down and then exhibited slurred speech, irrelevance and incoherence. He was expansive, euphoric and irresponsible in demeanour.

He was in rather a thin condition physically; but the general systems were clear, except for palpable, tortuous vessels and a blood pressure of 180 millimetres of mercury systolic and 100 diastolic. His pupils were unequal and irregular and did not react to light. Rombergism was present and his knee jerks were exaggerated and "floppy".

He was inoculated with malaria and began to lose weight rapidly. His lips became cyanotic. He had a few mild rigors, but was very restless and soon became weak. During the cold stage of one rigor he collapsed and lost power in his right arm. He then developed œdema of the face, which, however, soon subsided. After the completion of the treatment, his physical condition improved, and at times he was quieter and more coherent, although he was generally restless, noisy and elated, and often stripped himself. There was little change in his physical condition.

Tabes Dorsalis.

A female patient, suffering from *tabes dorsalis*, was shown. She was thirty-nine years of age. She gave birth to a still-born baby when she was seventeen. She married and had another pregnancy which also resulted in a still-birth. She stated that she was treated for meningitis when twenty years of age. She had been ill for some years before admission to hospital.

She complained chiefly of mental depression. Shortly before admission she experienced difficulty in walking. Three months before admission she had severe pains in the dorsal region, radiating to the front of the chest; later she had similar pains in the left leg. She said she felt as if she were walking on feathers.

At the time of admission she was found to be depressed and to have suicidal ideas, symptoms reactive to her physical state. The cranial, sensory and motor nerves were intact; the pupils reacted to light and accommodation. There was diminished sensibility to pain and heat in the legs, and pin-prick was interpreted as touch. There was also loss of sense of passive position and movement. There were localized areas of stinging, burning pain in the chest, and gripping pain around the head. There was hypotonia of the muscles, and the gait was ataxic; she walked on a wide base, turned unsteadily, and swayed when standing with her eyes closed. The knee jerks could not be elicited, and the plantar reflexes were flexor.

The blood serum reacted to the Wassermann test. The result of the Wassermann test of the cerebro-spinal fluid was "indefinite negative"; the cells numbered 400 per cubic millimetre of fluid; the globulin and Takata Ara tests were positive; there was no reaction to the bicolour guaiac test.

The patient was inoculated with malaria.

Psychogenic Mutism.

A man, aged forty-four years, was shown. He had been mute since November, 1932, when he was in Grafton Hospital for the extraction of teeth. He was anæsthetized for this operation, and on his recovery, he would not speak, and wandered aimlessly about the hospital. After a few weeks he refused his food, became threatening in manner, and attacked a nurse. He was removed to the police station and was certified as insane and sent to the Mental Hospital, Gladesville. The medical certificates were incomplete, however, and his discharge was arranged. There had been an attack of mutism, lasting three weeks, about seven months previously. He was unable to offer any explanation for his condition. He had led a lonely life in the country and had had to work very hard, but there had been no worries or shocks of any description.

At the time of admission he was quite mute, but replied to questions satisfactorily by writing his answers. He was bright, cheerful, alert and responsive. He indicated that his behaviour at Grafton was due to loss of control which he could not help. There was no evidence of any delusional or hallucinatory ideas. His physical condition was good, and there were no signs of any involvement of the cranial nerves. The reflexes were normal, and the blood did not react to the Wassermann test.

He remained almost entirely mute after admission, except for occasional monosyllabic answers. He smiled rather foolishly and evasively at times and, occasionally, was sullen and resentful of any interference. He was otherwise well conducted and amenable.

The next patient suffering from mutism was an unmarried woman, aged thirty-three years. She was the eldest of two living children; four had died in infancy. The father had alcoholic bouts till six years before her admission; since that time he had been a total abstainer; his father had had a similar history. The mother was "highly strung". The only brother was healthy. An aunt had been certified as insane for a brief period, and a first cousin had developed delusional insanity.

The patient had a healthy childhood. She was considered clever at school. She lived at home with her parents after leaving school. When younger, the patient broke off a friendship with a young man because of her attraction to someone eleven years her senior. They were friendly; but the man had never spoken of marriage, and the patient was shy about the subject. She was rather seriously minded and very religious. She never entered into social enjoyment, although she would work very hard for all the different fêtes and balls.

Two and a half years previously the patient had become suspicious and delusional, after a period of twelve months during which she had been sleepless and irritable. She expressed ideas of poisoning; and remarked that people were conspiring against her *et cetera*. She talked of suicide. After three months she improved, and then remained fairly well for nine months. She then became delusional again, careless and neglectful. She developed a stutter and her speech became unintelligible. After a time this improved, and she became able to speak quite well again. During the six months before admission she became mute. She was able to whisper "yes" or "no" at times. She took no interest in anything, and refused her food.

Examination revealed that she was poorly nourished, and had a very unhealthy skin. There was no evidence of any organic disorder. She was mute, unresponsive, and immobile, and maintained a rigid attitude. She rather resented attention and was easily moved to tears. There was no change in her condition after admission.

The third patient suffering from mutism was a girl, aged eighteen years, who came from Noumea. She had been quite well until the beginning of December, 1932, when she received word that she had failed in a school examination. She became very upset and delirious; she had pyrexia and was confined to bed for a fortnight. This state was diagnosed as meningitis. She subsequently became dull and disinterested, morose and silent.

She remained mute after admission. She was observed occasionally to laugh foolishly. She was passively resistant to ordinary attention, but actively resistant to bathing. She had a habit of tearing paper into small pieces. There was no indication of any organic disorder, and there was nothing to suggest that the patient was suffering from encephalitis. It appeared that the symptoms represented a schizoid type of reaction to the disappointing results of the examination. Her relatives stated that she had built up hopes of obtaining a good pass, as she desired to enter the teaching service.

It was remarked that, in view of the acute onset, there was some justification in giving a favourable prognosis as far as the present attack was concerned.

The fourth patient suffering from mutism was a woman, aged thirty-two years, who, since February, 1932, had been depressed and self-depreciating, regarding herself as hopelessly incompetent and unfitted to carry on her work as a school teacher. She complained of dysentery and insomnia, and took up light domestic work, believing that this was all she was suited for, although her head mistress was more than satisfied with her ability. She soon began to lose weight rapidly, although she could ill afford to do so. She became hopeless in outlook and felt that she was imposing on her friends and that she was entirely unworthy. She began to complain of persistent constipation, and would introduce her finger into the anal canal with the object at first of obtaining relief, but later on for the purpose of gratification, and eventually produced a condition of prolapse through this habit. Eventually her whole attention became centred on her bowels, and she believed that her food "did not pass through her".

There had been a previous breakdown in 1929, while she was studying abroad in England, France and Norway. Apparently she had been working hard, and had become very depressed and emotional. She consulted a psycho-

analyst, who stirred up a number of repressions without any particular benefit. It was during this period that she suffered from *prolapsus recti* and was operated on. In spite of the psycho-analytic treatment, she managed to recover from her melancholic state in about nine months, and seemed fairly well on her return to Australia. During her stay in England she joined a nude club, which was apparently not entirely idealistic in its objects. Her health had always been good, and she was very bright and happy in disposition, popular and friendly. Her mother had suffered from depressive psychosis and died in a mental hospital.

When admitted to Broughton Hall she was very depressed, self-accusatory and introspective. Her attention was almost entirely centred on her alimentary canal. She lacked confidence and initiative, and was hopeless in outlook, retarded, and indifferent to her environment. Her physical condition was very poor; she weighed only 31·5 kilograms (five stone), and her hair was very thin. The systolic blood pressure was 84 and the diastolic 66 millimetres of mercury. The other systems were clear and the tendon reflexes normal.

For a time after admission she showed some improvement, took her food well, and put on weight; but she remained self-depreciatory. She gradually became more seclusive and resistive, refused her food, and had to be spoon fed. Eventually she refused to speak at all, sitting by herself with her eyes closed. At the time of the meeting she was obstructive, obstinate and mute, and difficult with her food. It was pointed out that obstinacy, resistiveness, and sullenness were characteristic of the class of analerotics described by Freud. For more than five years the sexual instincts had been diverted to this infantile form of gratification, of which her present attitude was a temperamental expression. The joining of the nude club was probably an attempt to develop a more normal sexual relationship, which had been unsuccessful. The persistent self-depreciation and depression represented an emotional reaction to the situation which had developed as a result of her perversion.

Parkinson's Syndrome.

A male patient, aged twenty-six years, suffering from post-encephalitic Parkinsonism, was shown. In 1918 he had a severe attack of influenza, and almost died. For one week he had diplopia, and he was ill for six weeks altogether. He recovered from this and remained quite well until two years before admission, when he felt he was becoming very weak and unable to work; since then he had done nothing. Six months before the meeting "his nerves became bad"; he was tremulous, and his condition was such that he was certified as insane and sent to Gladesville Mental Hospital, where he remained for two months.

He was dull and disinterested on admission, exhibiting bradyphrenia and bradykinesis. His face was expressionless; but he was a great deal more alert than his appearance suggested. There was no evidence of any delusions or hallucinations at this time, although he was said to have been "imagining things" prior to admission.

Vision was normal; but his pupils reacted sluggishly to light and not at all to accommodation, and the left eye failed to converge. His face was smooth. His tongue was tremulous and his speech characteristically monotonous. General sensation remained normal. Rigidity was present in the right arm and leg, and this was "cog-wheel" in type. All his movements were slow, including his gait, in which there was very little movement of the right arm. Retropulsion and anteropulsion were present in a moderate degree. There was a slight fine tremor of the right arm. The tendon reflexes were increased on the right side. The condition was therefore hemiplegic in distribution. Except for a rather low blood pressure, there was nothing abnormal in the other systems.

In spite of increasing doses of tincture of stramonium, there was no objective improvement in his condition after admission, although he stated that he felt some benefit from it, in that his movements were freer. He had expressed hallucinations of sight and hearing, saying that voices continually talked to him about a variety of subjects, the nature of which he did not remember, and that he had seen snakes, horses and other things beside his bed at night. For a short period he was restless, foolish and irresponsible, wandering about the dormitory and chewing the stems of flowers. At the time of the meeting he was quiet, slow in speech and ideation, but amenable and quite bright and optimistic, in spite of his obvious disability.

The second patient suffering from Parkinson's syndrome was a married woman, aged thirty-three years. For the previous three years she had become increasingly dull and listless. She attempted to do her housework, but could manage it only with difficulty, because her movements were slower and she always felt languid and tired. This condition had become more pronounced in the previous six months. She did not know her age, but she could recollect events in the remote past very well. At times she was very emotional, and became rambling and irrelevant in speech, for a short period. Her husband noticed that her face looked "vacant" and her voice became monotonous. She stated that she had a severe attack of influenza in 1918, and was in bed for a fortnight. There was no history of any more recent pyrexia. She never had any serious illness, but had five operations on the frontal sinus for a cyst, said to contain a tooth; the last operation was just prior to the onset of the present trouble.

She was quite bright and cheerful on admission, but rather childish in manner. There was loss of memory, mostly for events in the previous three years. She did not know her own age, and actually gave it as thirty-eight, when it was only thirty-three, contrary to feminine custom. She said that her son was seven, whereas he was really ten, and she did not know the day, date, or month. Her attitude towards her condition was a passive one; there was indifference, amounting almost to emotional apathy. She tended to wander aimlessly about the ward. Her speech was slow and monotonous in quality.

Her general condition was satisfactory. Her face was expressionless, and the power of mimesis was lost. The pupils were equal and reacted normally to light and accommodation. The fundi were normal. Ocular convergence was good. There was a suggestion of muscular hypertonicity on the left side, and in walking the associated movements were absent on this side. Her gait was slow and rather shuffling, but retropulsion and anteropulsion were absent. Her reflexes were normal.

There was no change in her condition after admission. She remained quiet, regarded her condition with equanimity, and showed the same facial immobility. She was having increasing doses of *tinctura stramonii*, with subjective benefit. She stated that her energy had increased, but this was not obvious.

Post-Encephalitic Hemiplegia.

The next patient was a girl, aged nineteen years, who had been seen first by a medical practitioner in the country on June 8, 1930. She then had a complete flaccid paralysis of the right side, including the face, accompanied by aphasia. Apart from a cold for a few days, she had not been ill, and examination revealed nothing to account for the paralysis. There was no pyrexia, coma or dulness. The urine was clear and the blood pressure was normal. After three or four weeks she gradually regained the use of her right leg and was able to say a few words. In the absence of any other evidence as to the ætiology, and in view of the fact that she had, six months previously, complained of twitching in the left hand, her condition was regarded as probably hysterical. At the time of the meeting the patient had been in Broughton Hall for a period of four months.

At the time of admission the cranial sensory nerves were intact; there was paresis of the right side of the face. Speech was unintelligible, although she appeared to understand what was said to her. The right upper and lower extremities were paretic, and she walked with a hemiplegic gait. There was practically no power in the upper extremity. Sensation was diminished on the right side of the body. The knee jerks were hyperactive, the plantar reflexes were flexor. The tendon reflexes in the

right upper extremity were exaggerated. The mental state was characterized by dulness and lack of interest, indicating a moderate degree of general deterioration (dementia). The blood serum did not react to any of the tests for syphilis. The systolic blood pressure was 120, and the diastolic 100 millimetres of mercury. The urinary system was clear.

After admission there was no change in the condition of this patient. It had been noted that she wet the bed on several occasions.

The following comment on the case was made:

> There is nothing in the antecedent or family history of this girl to throw any light on the ætiology of her hemiplegia, apart from the paralytic symptoms themselves. There has been little evidence pointing to a gross involvement of the brain. Such evidence consists of the "cold" which preceded the onset of the paralysis, and a statement by the patient that, on the day preceding her illness, she had to leave work on account of feeling "sick all over". Nevertheless, the hemiplegic symptoms are of the organic type. It is suggested that they were precipitated by an encephalitis determining a thrombosis of the middle cerebral artery.

Secondary Dementia.

A patient suffering from secondary dementia was shown. She was a married woman, aged thirty-nine years, and had three children. Her husband was a farmer. One of her brothers had a slight breakdown during the bad times on the land. A sister had become depressed and mute, but had recovered from the attacks. The patient had been delicate as a child, and had suffered from bilious attacks. At eleven she was taken away from school, as she could not stand the confinement of the school room. She grew up strong, and had good health after her marriage. She was always active, and had a bright and happy disposition, but was retiring and very nervous in company. Her husband had had financial worry on account of drought and bush fires.

She became ill about two years before the meeting, after a lot of worry through two years of drought. She was very emotional, seemed to lose interest in things, and had to be fed nasally for four months. She recovered to a certain extent, but during the three months before admission had not been well. The main trouble was that she would not get out of bed. She complained that she was too tired. She neglected herself and children, and took no interest in the home.

She was rather sallow faced, but moderately well nourished. The systolic blood pressure was 115, and the diastolic 80 millimetres of mercury. The general systems were clear and the menses regular.

She was able to recall past events correctly, but was indifferent to recent happenings and seemed to be very confused as to her illness. She was dull, rather foolish, and self-effacing, was childishly satisfied, and had to be prompted to do things. She was careless and untidy.

At the time of the meeting the patient had been in hospital for six months. Her condition seemed to be deteriorating. She was often found in bed smiling vaguely during the day. Recently she had been drawing pictures of trees, all of the same stereotyped design.

Anxiety Melancholia.

A male patient, aged forty-three years, suffering from anxiety melancholia, was shown. Three years before the meeting his son had died after a short illness, and at the same time the patient lost his position as a motor body builder. He began to worry after this, and gradually became more depressed and hopeless in outlook. He was upset by trifling matters, such as a slight inaccuracy in his income tax returns, and distressed himself unnecessarily. He worked as a car-minder until the police ordered him off the streets about two months before the meeting. From then onwards he became more miserable and agitated, pulling his hair out, and he became restless and apprehensive, particularly at night. He became reserved, solitary and moody, and began to think he could not trust anybody. He admitted that he worried because he had some arsenic and lead in his possession for the purpose of exterminating white ants, and he was afraid that the poisons might cause harm to someone and he would be blamed. There was a history of a similar attack, but of a less severe nature, some twelve years previously. His previous health had always been good, and his loss of employment was due to the firm's failure, and was no fault of his. His father had died in Parramatta Mental Hospital some years previously, and this influenced him considerably.

He was depressed, restless and somewhat agitated on admission, and was unwilling to discuss his difficulties, being afraid that his statements might be held against him. He was self-depreciating, anxious and tremulous, and apprehensive that some harm would befall him. He continually tossed about in bed, rubbing his head and wringing his hands. His tendon reflexes were normal and his pupils reacted to light and accommodation. The systolic blood pressure was 180 and the diastolic 100 millimetres of mercury. The rise was probably due to his extreme agitation.

After his admission he improved considerably. It was remarked that the condition was the result of several difficulties which had arisen about the same time: grief at the loss of a son, unemployment, financial troubles, and a tendency to a depressive state of mind, all of which had combined to produce an acute anxiety state. The rest and absence from a worrying environment had helped to ameliorate his condition, and, at the time of the meeting, he was much brighter and less anxious about the future, although he complained that he had difficulty in remembering names and incidents, a condition that was probably due to a failure of concentration and some residual mental instability.

Congenital Mental Deficiency with Rhachitic Dwarfism.

A single woman, aged twenty-five years, was shown. She was the third of five children, two of whom were quite healthy. The second child had been educated in the deaf and dumb institute and had been sick for five years with tuberculous disease or hydatid lung abscess, and had died at the age of twenty years. The youngest child had a birth palsy of the left arm and slight curvature of the spine. She was mentally alert. The patient's parents were healthy. The patient was delicate at birth, and had hydrocephalus and rickets. She took infantile convulsions until the age of three years. In her early attempts at walking she was apt to stumble, and she did not talk until the age of five years. From then on she had been treated with thyreoid extract. She had contracted measles and whooping cough in childhood. She was backward at school and was unable to get past the third class. She had special tuition in needlework and music, but could not make any progress at the latter. She could sew very well, did light domestic duties, and was neat and clean in her work. She was cheerful, and very friendly and affectionate; at times she was a little bad tempered, but was very easy to manage. The menarche was established at the age of fifteen years. The menses were scanty, but regular.

Two months before admission she went for a holiday, and, while away, she took too many thyreoid tablets. At the time it was thought that she was suffering from thyreoid intoxication, and she was treated for such by the local medical practitioner. Since then she had become restless and unhappy. She complained of head noises and burning sensations. She was unsettled, and wanted to leave home, and was emotional and somewhat obstinate.

Examination revealed that her growth was stunted and her nutrition fair. She had a large head and a broad, square forehead, with prominent frontal eminences. The chest was long, and the sternum was depressed; there was a well marked Harrison's sulcus. The costal border of the ribs was prominent and everted. The abdomen was pot-bellied. The arms were well developed, but the legs were deformed. She was knock-kneed, and there was a bowing out of the upper one-third of the tibia; there was a bony prominence at the head of the fibula, possibly enlarged epiphysis. The face was somewhat flattened, and hollow about the orbits. The lower jaw protruded and overlapped the upper; the patient was edentulous.

The tongue was large and deeply fissured. There was slight tachycardia. There was huskiness of speech. The gait was stumbling. The patient tilted her pelvis as she walked.

The mental state was that of a child aged about seven or eight years. She was miserable and hypochondriacal.

At the time of the meeting, the patient had been in hospital for four months. There had been no marked improvement. She was restless and very unstable, was inclined to be somewhat obstructive at times, and cried noisily if she could not have her own way. She complained of aches and pains, and took no interest outside herself. She was very mischievous in the ward.

Puerperal Psychosis.

A married woman, aged thirty-six years, was shown. She was the eldest but one in a family of thirteen. She had two sisters of a nervous temperament. Her father had one sister an epileptic, and another sister who was certified insane at the menopause. The father's brother died during a mental breakdown, and one of his nephews committed suicide.

The patient had a healthy childhood until the menarche, which was delayed. The menses ceased for six months after the first two periods. At school the patient was backward. She assisted her mother at home until her marriage at the age of twenty-eight years. She had three children: a boy, aged five years, a daughter, aged three years, and a son, aged six weeks. She had always been rather quiet and never went out very much.

She was well for two weeks after the baby was born, but during the month prior to admission she became depressed. She found it difficult to manage the house work and began to worry about past wrong-doings.

Her physical condition was fair. Examination revealed no evidence of any physical disorder. She was depressed, retarded and very self-accusatory. She was probably normally of a dull intelligence. She was quiet, disinterested, and absorbed in her ideas of wrong-doing. After admission she became more depressed and retarded. She expressed delusions of sin and was very self-accusatory. She spent most of her time praying. She was pessimistic in outlook, was somewhat resistive, and had to be spoon fed.

The second woman suffering from puerperal psychosis was thirty-seven years of age, and had three children. She was the elder in a family of two. Her mother died in Callan Park Mental Hospital of melancholia at the menopause. Her father suffered from asthma for years. The patient had a healthy childhood. On leaving school she kept house for her father. She was always very shy. She married happily and had three children, the eldest of which was eight years and the youngest six months.

About two months after the birth of the third child she seemed to collapse. She felt terribly weak and did not have the strength to do her house work. She became very unhappy and worried about the children. She was constipated and had ideas that her bowels would not act. She thought that the doctors could not do anything for her.

Her physical condition was satisfactory. She was very depressed, worried, and hopeless in outlook, and had ideas of somatic derangement. She was miserable and emotional, vaguely anxious, and unable to express her worries. Very little improvement occurred after her admission.

The third patient suffering from puerperal psychosis was thirty-four years of age. She had two children, the younger two months old. She was the fourth in a family of ten. Her parents were healthy; but one sister had fits of mild despondency. The patient herself had always been healthy. From the age of fifteen until her marriage she was employed as a machinist in a work room. She was good at her work. She had been unable to suckle either of her babies. Fourteen months before admission she had a miscarriage at six months. She was always very energetic, and was impatient and worried over trifles. She had not had very much pleasure and was not fond of going out. Her husband did not have regular work.

Her confinement was normal. Two weeks later she began to feel depressed and to lose interest; she became very much worse. She said that she was "done" and that there was no hope for her. She also made several suicidal attempts just prior to admission.

Examination revealed no evidence of any physical disorder.

She was very depressed and self-accusatory. She expressed the idea that she had lost her soul and brought harm to her family. She had no insight into her condition, and had very active suicidal tendencies. She was very unsettled and was resistive with food and medicine.

After admission she became very restless and confused and had to be put in a single room. She climbed out through the fanlight to the veranda roof, and jumped off, causing severe abrasions. One week later she was found with a sheet tied tightly round her neck; her face was discoloured and her lips puffy. She was very restless and agitated for a time, and said that she was responsible for the deaths of all the patients in hospital. At the time of the meeting she was not quite so restless, but was somewhat confused; her habits were very dirty. She was difficult with food, and, as a consequence, was very thin. She also had a habit of picking at her face and finger nails.

The fourth patient suffering from puerperal psychosis was thirty-eight years of age and had five children, the youngest four months old. She was the fourth in a family of twelve. She was born in England and came to Australia at the age of seventeen years. One brother committed suicide through a love affair at the age of twenty-one years. She could give no account of her father's people; otherwise the family history was clear. She was a backward scholar, and, from the age of twelve years, worked at lace making, and, later, in Sydney, in domestic service. The most she earned was sixteen shillings a week. The patient was unable to suckle any of the children, owing to the failure of milk secretion. She had a comfortable home until eighteen months before admission. Her husband had been unemployed for twelve months. One month before the confinement he went to the country for work. The patient had not previously been alone.

The patient had felt weak ever since the last confinement. She noticed that she perspired a lot. She felt she could not be bothered doing the house work. One month before admission she began to feel miserable. She became frightened that she might harm the children and herself. She would leave the home to get away from this feeling. She was troubled by words and sentences that were being repeated in her mind, that she was going to die, or that she was going mad *et cetera*. She slept little, and had a dull feeling on the top of her head.

The patient was somewhat under-nourished, and appeared physically exhausted. She was depressed, anxious and apprehensive.

After admission she became much more settled, and at the time of the meeting she was able to sleep, and, although she still complained of the talking that was going on in her mind, she said it was much less severe. She was more hopeful in outlook and less anxious about herself.

The next patient was a woman, aged twenty-eight years, who had one child, aged three months. She was the elder of two children. Her mother was of a nervous temperament, but her sister was healthy, and the family history was clear. The patient was well cared for as a child, and attended school until she was fourteen years old. She was an average scholar. For six years she worked on a power machine in a factory, and had charge of fifteen girls when she left to be married at the age of twenty-two years. Three years before admission she had a still-born premature baby. She was happily married. Three years before admission her husband lost constant work and had been unemployed for a period of six months. The patient was always rather quiet and somewhat backward in meeting people.

The patient had been ill for five months before admission. She became sleepless two months before the confinement, was worried and afraid the baby would be born with a birth-mark. After the birth, the patient worried that the baby would be affected by her (the patient's) state of mind. She said she was not normal. She appeared very distressed and unhappy, and seemed

to take no interest in the baby. She was unable to carry on her household responsibilities.

At the time of admission her physical condition was satisfactory. There was a history of two weeks' insomnia following still-birth three years previously.

The patient talked readily about herself, and was mildly depressed and disinterested. She was morbidly introspective and preoccupied with the idea that she was mentally affected, also that her outlook was hopeless. She was distressed by her state, was emotional, and had vague ideas of poisoning herself.

The patient remained quiet and amenable after admission. She appeared a little brighter, although she was unhappy and emotional at times. She was mildly disinterested and doubtful about her recovery.

The last patient suffering from puerperal psychosis was aged thirty-five years, and had five children. She was born in Dorset, England, and came to Australia at the age of twenty-five years. Her father committed suicide; he had been a heavy drinker. Her mother died of a malignant goitre; her sister had a goitre; this disorder was common in the district where the family had lived. The patient had a simple enlargement of the thyreoid from the age of twelve years; she had been healthy otherwise. She worked as a domestic servant, her husband being a labourer. He worried her on account of his drinking habits and was apt to be abusive. The eldest child was nine and the youngest two weeks. Five years before admission thyreoidectomy was performed on the patient; she was worried on account of her mother's history. Shortly after the operation she became pregnant.

She had been admitted to Broughton Hall seven years previously, but had only stayed a few days, and her condition was diagnosed as anxiety hysteria. Three years later she was readmitted, having been ill for twelve months. After eight months she was discharged recovered. Her condition again appeared to be one of anxiety hysteria.

She had been confined two weeks before admission. Two days later she became worried and delusional, and expressed the idea that people were talking and laughing about her, and that wireless messages told everybody that she had a disease. She was depressed and worried. She was in a poor condition and looked exhausted. There was no evidence of lactation, and she had a slight vaginal discharge. The systolic blood pressure was low and her pulse was small and feeble. She was depressed and apprehensive, and somewhat confused. She could hear people talking about her, and expressed persecutory delusions.

After admission she became very restless and worried. She was very disturbed by her ideas and her fears for the safety of her children, and believed her baby was dead. For a time she expressed ideas of being tortured. She pleaded not to be tied up in a bag with snakes *et cetera*, and thought she was to be sent away. She was talkative about her ideas, and noisy and disturbing at night. She took her food poorly.

Pernicious Anæmia.

A married woman, aged sixty-one years, was shown. She had an adult family of seven, and had generally enjoyed good health. She was first admitted to Broughton Hall three years previously, when she stated that she had been ill for five years. For a month she was troubled with attacks of "hives" on the thighs. She then went to the Royal Prince Alfred Hospital out-patients' department, and was treated for "nerves", and was eventually admitted to Broughton Hall with a diagnosis of neurasthenia. She complained that for the previous nine months she had had "pins and needles", first in the feet, then in the legs, sometimes also across the abdomen. She suffered from cramps in the legs and loss of feeling, and noticed a weakness on going up stairs. Two weeks before admission she fell down twice in the street.

Physical examination revealed some disturbance of sensation in the lower limbs. The sensation of pin-prick was dulled, and there were areas of patchy analgesia. There were some hypotonia and weakness in the legs. For five weeks after admission she continued to complain of cramps in the legs, weakness and "pins and needles" in the left arm, and sensations of coldness, also pains across the abdomen.

Examination of the blood revealed that there were 2,500,000 red blood cells per cubic millimetre, that the hæmoglobin content was 65% and the colour index 1·28. The white cells numbered 3,437 per cubic millimetre, made up as follows: neutrophile cells, 29%; basophile cells, 17%; lymphocytes, 70%. In a second blood examination the red blood cells were seen to be large, and two megaloblasts were counted. A test meal showed no abnormality in the gastric secretion. After two months of liver treatment the blood picture became normal. She was discharged from hospital after four months.

At the time of readmission she stated that she had been well until three months before. She had not taken the liver consistently, and recently had not taken any. She noticed a "deadly weakness in her legs", and her knees began to give way. She developed pains in the hips, her abdomen became swollen and her bowels obstinately constipated. She had not walked for three months before readmission.

Her abdomen was very prominent, full and tense; the abdominal reflexes were absent, the abdominal muscles were hypotonic, and she had retention of urine. She had some disturbance of articulation. She was unable to walk, and was paraplegic. There was a marked hypotonia, the knee jerks were absent, and Babinski's sign was marked on the right and slight on the left. There was also a pronounced sensory change in the lower extremities. The pin-prick was felt only as touch, and there was retardation of sense of touch and muscle sense. She had neuritic pains in her legs and hips, and areas of hyperæsthesia. She had some paresis of the right ear, and the reflexes were not as brisk as those of the left arm. There were 3,975,000 red blood cells per cubic millimetre of blood, the hæmoglobin value was 80%, and the colour index 1·0. There were 6,500 white blood cells per cubic millimetre. There was a moderate degree of anisocytosis, and there was a predominance of microcytes. No free hydrochloric acid was found in the stomach after a test meal.

For the first two weeks after admission the patient had to be catheterized. Large doses of purgatives were still required at the time of the meeting. There was no improvement in her legs or right hand; but the neuritic pains in the hips seemed less severe. She was having liver treatment.

The next patient suffering from pernicious anæmia was thirty-three years old; she had three children. Her mother had died in the Mental Hospital, Kenmore. The other members of the family were healthy. The patient's husband had a small property. They had been "up against it" for the previous six months. The patient had always been in good health.

She had not been well since the last baby was born two years before admission. She was anæmic before the confinement, and had no milk for the baby. After the confinement she was treated for six weeks for weakness. Her health was indifferent for four months. When she came to Sydney she was treated for anæmia. She returned to the country and was well for seven months; but during the fourth months prior to admission she felt that she could not keep going. She complained of weakness and was unable to work. It was only in the previous few weeks that she developed any mental symptoms. She seemed to become depressed and restless, and expressed ideas that harm would come to the family.

She was depressed and very agitated on admission. She was retarded and preoccupied with persecutory ideas. She was unsettled and sleepless.

The patient had a pale, anæmic appearance. Her blood pressure was rather low.

At the time of the meeting she had been in hospital just over two months. There was a marked improvement in her mental state. She was no longer depressed, and lay quietly in bed. She had begun to interest herself in reading. One month before the meeting there were found to be 4,250,000 red blood cells per cubic millimetre, the colour index was 1·0, and the hæmoglobin value 98%. Examination after the administration of a test meal revealed a complete absence of free hydrochloric acid in the gastric secretions; there were only twelve cubic centimetres of free acid one hour after the meal.

(To be continued.)

NOMINATIONS AND ELECTIONS.

THE undermentioned has been elected a member of the New South Wales Branch of the British Medical Association:

Hewitt, Lance Ellis, M.B., B.S., 1930 (Univ. Sydney), Singleton.

The undermentioned has been nominated for reelection as a member of the New South Wales Branch of the British Medical Association:

Bennetts, Harold Graves, M.B., M.S., 1896 (Univ. Sydney), D.P.H.R.C.P.S., 1907 (England), F.R.C.S., 1907 (Edinburgh), 201, Macquarie Street, Sydney.

Correspondence.

DIATHERMY OF TONSILS.

SIR: Dr. Bettington should be able to stand up to a little friendly criticism without reacting in the manner he does. My remarks were not intended to be taken in the personal way the tone of his letter discloses, but only to deny his statements that the operation of diathermy of tonsils is painful and the results unsatisfactory. I stated that I had done about 300 cases, and Dr. Bettington, in his reply, says that his experience is not far behind mine. It seems an extraordinary thing to me that he should put nearly 300 people to torture and achieve unsatisfactory results, before suddenly deciding to give up the method. The fear which he accuses me of must have been a nightmare to him during the whole time he was using electro-coagulation.

Unlike his contemporaries, he did not find any alarming hæmorrhage, nor cause any peritonsillar damage, such as that described by one surgeon, who published in this journal a diagrammatic sketch showing the anterior pillar having first been removed in order to get at the tonsil. His chief objection is pain during and after the operation, and I think he was wise to discontinue the method. He also disparages diathermy because he regards it as difficult to remove the whole tonsil. It is strange that tissue (infected and malignant tissue as well) can be removed from all parts of the body by electro-coagulation, and a healthy scar result, but not so lymphoid tissue of the throat. I can only reiterate that I do not find patients complain of severe pain during or after the operation and that I believe the destruction of tonsillar tissue is just as practicable as elsewhere in the body. Even if a suitable non-inflammable general anæsthetic were necessary, I would still regard this method as the safest and most satisfactory.

As for Dr. Ashleigh D. Davy, who ventures into the controversy, I have little to say, as he does not offer any destructive criticism of diathermy. He seems amazed that 100% results could be obtained by any method. Now, I have always considered that gentlemen practising throat work as a specialty were in the position to promise complete removal of tonsillar tissue by dissection in 100% of cases; but at least one is to be found who not only disclaims such a guarantee, but is astonished that it should be considered in the realm of possibility. He would do well to keep to mathematics and leave diathermy of tonsils alone.

Yours, etc.,

F. WILKIE SMITH.

Rosebery,
April 28, 1933.

THE NEW SOUTH WALES MEDICAL BOARD.

SIR: I have felt that being registered as a legally qualified medical practitioner and in varying degree controlled by the General Medical Council at Home or by the State Medical Board in Australia was at least some protection to me and to those of my professional brethren who try to do honest work, and, in doing so, do not hesitate to give opinions that may not be acceptable to the "powers that be". But I begin to wonder what may happen, when I find that a Minister for Public Works and Health, or, indeed, any other Minister, should have the power to abolish or appoint a judicial body such as the New South Wales Medical Board. Even though he were "not actuated by caprice or whim or fancy", that he should have such power would not in these days of democracy be credited, any more than that a Minister for Justice, or any other Minister, should have the power to interfere with the Supreme Court that controls the education, registration, and deregistration of members of the legal profession. A thoughtful member of the public would rightly maintain that such a power is contrary to the public interest, to exercise it may be an abuse, and it should not be vested in any one political personage, even though he might handle the matter like a super-statesman, "graced with polish'd manners and fine sense", and not wanting sensibility. Yet such, unfortunately, is still the state of the law in New South Wales as regards the Medical Board (*vide* THE MEDICAL JOURNAL OF AUSTRALIA, page 389, March 25, 1933).

In Great Britain the body corresponding to the Medical Board is the General Medical Council, which body is established by Parliament as a body corporate, having perpetual succession and a common seal, with power to hold lands. It consists of many members, namely: (i) Persons "nominated" from time to time by His Majesty, with the advice of the Privy Council; (ii) persons "chosen" from time to time by the universities and other medical corporations of England, Ireland, Scotland and Wales; (iii) persons "elected" from time to time by registered medical practitioners resident in those countries and called direct representatives.

Election is by postal voting; vacancies are filled as they occur from time to time. A Direct Representative is elected to hold office for the term of five years, and is capable of being reelected; the Council elect one of their members to be President. Such a body as the General Medical Council has perpetual existence, and cannot be abolished except by Parliament.

It is high time (or is it past high time?) that the supreme court of the Medical Profession in New South Wales should be so equitably constituted, and so firmly established by statute, that no Minister or Ministry could interfere with such a body, except by reference to, and express consent of, the members of both Houses of the State Legislature in Parliament assembled.

If ever the Commonwealth, with the consent of the States, is given power to appoint a Registration Board or General Medical Council, its proposed form of constitution should be very carefully watched.

Yours, etc.,

R. H. FETHERSTON.

Melbourne,
April 30, 1933.

COAL MINER'S LUNG.

SIR: In the journal of April 29 there appears an article by Dr. Badham, industrial hygienist, and Dr. Taylor, analyst, on coal miner's lung, in which they severely criticize Australian radiography in pulmonary fibrosis. Their severe criticism is based on a study of thirty-six cases only.

I would like to hear the authors' opinion after a study of a further few thousand cases, and, if they continue this investigation, I would suggest that an expert radiologist be employed to interpret the skiagrams; in a recent series of coal miners examined at Waterfall, this course was not followed, and I am afraid the interpretation of skiagrams was not of the highest standard.

The one skiagram reproduced in the article is that of Case XII (one of the Waterfall series, I believe), and the radiological report on it is far from an accurate one. For example, the heart is described as being centrally situated, but it is really normal in position, while the description

of the lung appearances is far astray. Of course, if this is a sample of the radiographic interpretation to which the authors have been accustomed, I do not wonder at the severity of their criticism.

Now, the authors to the contrary, there has been extensive *post mortem* checking of the radiographic findings in pulmonary fibrosis, both in the Sydney Hospital, where I have practised radiography for twenty-five years, and at Broken Hill, and the percentage of correctness of interpretation has been found to be high in the nineties. Dr. George, of Broken Hill, could probably give exact figures in hundreds of cases seen by him.

There should be no difficulty to the expert in determining the presence of a superadded apical tuberculosis in a case of pneumonokoniosis, nor should there be any difficulty in distinguishing an apical tuberculous consolidation from that peculiar subpleural massive consolidation which occurs in coal miners, especially in men who have mined in Great Britain, and which seems to have given Dr. Badham such trouble. Basal tuberculosis is certainly a very difficult condition to recognize in skiagrams, whether pneumonokoniosis is present or not.

The authors state that the history of the man should be known before interpreting the skiagrams: I think too strict adherence to this rule is the cause of most of the misinterpretation met with, as the unskilled man is liable to colour his X ray report to suit this history. The X ray picture is one of projected pathology; it takes many years, and a study of many thousands of cases, before proficiency is attained in its interpretation.

As I am not an all-round specialist, I do not feel competent to express an opinion on the *post mortem* pathology of these lungs, but would respectfully suggest that Dr. Badham's "cavities" are not really pulmonary cavities, but are due to multiple bronchiectases, which are so common in old bronchitics with pulmonary fibrosis.

Yours, etc.,

J. G. EDWARDS.

185, Macquarie Street,
Sydney,
May 4, 1933.

MINERS' NYSTAGMUS.

SIR: In reply to the letter of Dr. A. E. Taylor, of Dandenong, Victoria, which was published in THE MEDICAL JOURNAL OF AUSTRALIA on May 6, 1933, regarding the question of miners' nystagmus.

Dr. Taylor suggests that the condition is not caused by defective illumination, but rather, that it is due to chronic gas poisoning, for example, carbon monoxide or some such poisonous gas. This suggestion was very carefully considered by the Medical Research Council in its first report on miners' nystgamus, and I would refer Dr. Taylor to the article on mine gases by Professor Haldane, published in this first report. Various experiments were carried out with carbon dioxide, carbon monoxide, fire damp (methane) and other mine gases. The proportion of carbon monoxide commonly present in mine air was seldom found to be over 0·005%, and this proportion was stated to have no appreciable physiological action. Professor Haldane completed his experiments by stating: "we may therefore conclude with complete certainty that the abnormal constituents in ordinary mine air have nothing whatever to do with the production of miners' nystagmus". Carbon monoxide can produce nystagmus, but if Dr. Taylor has seen any such cases he will realize that the oscillations of the eyeballs are horizontal in type and quite different from the rotatory oscillations seen in cases of miners' nystagmus. The generally accepted opinion is that defective illumination is the real cause of miners' nystagmus, and my experience in connexion with these cases is in accordance with this view.

Dr. Taylor's remarks about the pit lamps used in the Victorian mines only tends to emphasize the clinical facts which have been demonstrated repeatedly by ophthalmic surgeons who have had dealings with many cases of miners' nystagmus. I think, however, the conclusions which Dr. Taylor has drawn have been erroneous. We rarely, if ever, find nystagmus cases in naked light pits, and this is not because the mines are free from gas, but because the illumination from the naked lights, which are similar to those which Dr. Taylor describes, is much superior to the oil safety lamps and electric lamps. This fact can be verified in any naked light pit by the comparison between the candle power of the three types of lamps. The electric safety lamps at present in use vary from 1·5 to 2·0 candlepower at the commencement of the shift, and diminish towards the end of the shift. The oil safety lamps (Patterson and Dain type "Bifold") 0·6 candlepower at first part of shift to 0·25 candlepower at the end of the shift. Naked lights vary from four to five candlepower. The illumination from the naked light can be varied by the miner himself when working at the coal face, the wick being lengthened if necessary, giving a better illumination. Further, the lamp is worn on the miner's cap and directly illuminates the coal face on which he is working. The luminosity is fairly constant with the naked light, whereas with the oil safety lamp or electric lamp, the candlepower varies considerably and coal dust particles and moisture settle on the glass and fog the illumination. The lamps are often many feet away from the coal face with shadows cast by the lamp and by the miner working between the lights and the coal face. The naked lights are not ideal, and there are many factors in the working of a mine which render these lights unsuitable; but they are infinitely superior to any oil safety lamp, and, up till the present time, the electric lamps have not been sufficiently improved to give a satisfactory illumination.

I have examined many cases of miners' nystagmus in the Northern coal fields during the past fifteen years, and I have only seen two cases of nystagmus in the naked light pits, and this, I think, has been the experience of most other ophthalmic surgeons who have had dealings with these cases.

Dr. Taylor suggests it is impossible to imagine worse lighting than that which is present in the naked light pits of Gippsland. The naked lights used in the New South Wales coal mines are of a similar type to those which he describes, and yet these naked light pits are illuminated workshops compared with some of the oil safety lamp mines. One has only to compare a naked oil light with an oil safety lamp whilst the miners are at work to realize the part which defective illumination plays in the causation of this industrial malady.

Yours, etc.,

HUGH G. ALLEN.

Newcastle,
May 6, 1933.

Obituary.

ARTHUR HILL MURRAY.

WE regret to announce the death of Dr. Arthur Hill Murray, which occurred on May 4, 1933, at Cunnamulla, Queensland.

Books Received.

FOOD, HEALTH, VITAMINS, by R. H. A. Plimmer, D.Sc., and V. G. Plimmer; Fifth Edition; 1932. London: Longmans, Green and Company. Crown 8vo., pp. 165. Price: 5s. 3d. net.

DISEASES OF THE HEART, DESCRIBED FOR PRACTITIONERS AND STUDENTS, by Sir Thomas Lewis, C.B.E., F.R.S., M.D., D.Sc., LL.D., F.R.C.P.; 1933. London: Macmillan and Company, Limited. Royal 8vo., pp. 317, with illustrations. Price: 12s. 6d. net.

INTERNAL DERANGEMENTS OF THE KNEE-JOINT: THEIR PATHOLOGY AND TREATMENT BY MODERN METHODS, by A. G. T. Fisher, M.B., Ch.B., F.R.C.S.; Second Edition; 1933. London: H. K. Lewis and Company, Limited. Demy 8vo., pp. 219, with 120 illustrations. Price: 15s. net.

THE COMMON CAUSES OF CHRONIC INDIGESTION: DIFFERENTIAL DIAGNOSIS AND TREATMENT, by T. C. Hunt, B.A., D.M., M.R.C.P.; 1933. London: Baillière, Tindall and Cox. Crown 8vo., pp. 348, with 16 plates. Price: 12s. 6d. net.

PSYCHOLOGY OF SEX: A MANUAL FOR STUDENTS, by Havelock Ellis; 1933. London: William Heinemann (Medical Boks) Limited. Demy 8vo., pp. 344. Price: 12s. 6d. net.

Diary for the Month.

MAY 23.—New South Wales Branch, B.M.A.: Medical Politics Committee.
MAY 24.—Victorian Branch, B.M.A.: Council.
MAY 25.—South Australian Branch, B.M.A.: Branch.
MAY 25.—New South Wales Branch, B.M.A.: Branch.
MAY 26.—Queensland Branch, B.M.A.: Council.
JUNE 1.—South Australian Branch, B.M.A.: Council.
JUNE 2.—Queensland Branch, B.M.A.: Bancroft Memorial Lecture.
JUNE 7.—Victorian Branch, B.M.A.: Branch.
JUNE 7.—Western Australian Branch, B.M.A.: Council.
JUNE 9.—Queensland Branch, B.M.A.: Council.
JUNE 13.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
JUNE 20.—New South Wales Branch, B.M.A.: Ethics Committee.

Medical Appointments.

Dr. F. H. Beare (B.M.A.) has been appointed a Member of the Advisory Committee of the Mareeba Babies' Hospital, South Australia.

* * *

Dr. H. T. Bourne (B.M.A.) has been appointed Acting Medical Superintendent of the Hospital for the Insane, Kew, Victoria, pursuant to the provisions of the *Lunacy Act*, 1928.

* * *

Dr. E. F. Gartrell (B.M.A.) has been appointed temporary Honorary Assistant Physician at the Adelaide Hospital, South Australia.

* * *

Dr. D. L. Barlow (B.M.A.) has been appointed temporary Honorary Medical Officer in Charge of the Vaccine Clinic, Adelaide Hospital, South Australia.

* * *

Dr. M. Schneider (B.M.A.) has been appointed temporary Honorary Surgeon for Ear, Nose and Throat to the Parkside Mental Hospital, South Australia.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages xiv, xv, xvii.

CHILDREN'S HOSPITAL (INCORPORATED), PERTH, WESTERN AUSTRALIA: Junior Resident Medical Officers (male).
DEPARTMENT OF INSPECTOR-GENERAL OF HOSPITALS, ADELAIDE, SOUTH AUSTRALIA: Honorary Officers.
GRESSWELL SANATORIUM, MONT PARK, VICTORIA: Resident Medical Officer.
HOBART PUBLIC HOSPITAL, HOBART, TASMANIA: Junior Resident Medical Officer.
HOMŒOPATHIC HOSPITAL, MELBOURNE, VICTORIA: Resident Medical Officers.
LAUNCESTON PUBLIC HOSPITAL, LAUNCESTON, TASMANIA: Resident Medical Officer (male).
MATER MISERICORDIÆ CHILDREN'S HOSPITAL, BRISBANE, QUEENSLAND: Resident Medical Officer.
MOTHERS' AND BABIES' HEALTH ASSOCIATION (INCORPORATED), ADELAIDE, SOUTH AUSTRALIA: Honorary Medical Officer.
THE PUBLIC SERVICE BOARD, NEW SOUTH WALES: Medical Officer (male).
THE RACHEL FORSTER HOSPITAL FOR WOMEN AND CHILDREN, REDFERN, NEW SOUTH WALES: Honorary Medical Officers.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associated Friendly Societies' Medical Institute. Chilliagoe Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their agreement to the Council before signing. Lower Burdekin District Hospital, Ayr.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	Combined Friendly Societies, Clarendon and Kangarilla districts. All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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